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Original Communications

PROTECTION OF THE HEART BY ARTERIALIZATION OF THE CORONARY SINUS

I. CORONARY COLLATERAL FLOW IN NORMAL DOGS AND IN DOGS HAVING HAD PREVIOUS NONDESCRIPT CARDIAC SURGERY

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SURGICAL therapy for the treatment of coronary artery disease has been experimented with for over fifty years. Pratt[1] first suggested the possibility of maintaining the nutrition of the heart by reversing the flow of blood in the cardiac veins in 1898. He forced defibrinated arterial blood into the coronary sinus of freshly extirpated cats' hearts, and noted that both ventricles continued to contract in a regular and coordinated manner at intermittent periods for one and one-half hours.

Roberts and associates[2] created an arteriovenous anastomosis between a systemic artery and the coronary sinus in dogs by means of a connecting glass cannula. The cardiac veins became distended, pulsatile, and arterial in color. After it was felt that reversal of flow had occurred, coronary arterial ligation was performed. The hearts continued to beat regularly and forcefully for as long as twenty-six hours. Chicago blue dye injected into the coronary sinus resulted in complete delineation of the capillaries. The authors suggested that an ischemic myocardium may be revascularized by anastomosis of a large artery to the coronary sinus.

Beck and co-workers[3, 4] devised a procedure for the revascularization of the human heart in occlusive disease of the coronary arteries. The technique utilized the placement of a free vein graft between the aorta and the coronary sinus, following which the coronary sinus was partially ligated several weeks later. These workers have demonstrated that arterialization of the coronary sinus

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provides for adequate protection to the heart during major coronary artery ligation. Ligation of the left anterior descending coronary artery at its origin in normal dogs results in a 70 per cent mortality. Ligation of this vessel following arterialization of the coronary sinus results in a reduction of the mortality rate of 20 to 30 per cent. In addition, normal dogs surviving ligation of the left anterior descending artery present large myocardial infarcts. In the dogs with aorticocoronary sinus anastomoses surviving anterior descending ligation, myocardial infarcts are either small or nonexistent. Hahn and associates[5-7] were able to prepare three sinus-arterialized dogs which survived ligation of all coronary arteries except for the septal branch of the left coronary artery for a period of five months, at which time they were sacrificed. These workers also noted that the blood flowing in retrograde fashion from the distal cut end of the divided left circumflex coronary artery in such dogs increased as progressive occlusion of the left main coronary artery was performed. India ink injected into the graft was identified in the blood obtained from the retrograde flow of the divided coronary artery.

Smith and co-workers[8] attempted to elucidate the mechanisms of protection afforded by arterialization of the coronary sinus in acute experiments. They demonstrated that elevation of the coronary sinus pressure without arterialization reduced the mortality following circumflex coronary artery ligation from 70 per cent in normal dogs to 40 per cent. Coronary sinus pressure elevation with arterialization of the sinus in these acute experiments further reduced the mortality to zero. They concluded that arterialization of the sinus provided the greatest protection against ventricular fibrillation following coronary artery ligation, and that such arterialization was the only practical means of chronically elevating coronary sinus pressure to effective levels.

Eckstein and associates,[9, 10] in a series of acute experiments, stated that a small retrograde flow was present in dogs in which the coronary sinus was arterialized, using a glass cannula for the anastomosis. They noted that the retrograde flow from the distal segment of the occluded circumflex artery apparently increased with augmentation of pressure in the coronary sinus, and was related to the diastolic pressure.

In recent years, we have been interested in arterialization of the coronary sinus for the surgical therapy of occlusive coronary artery disease. Bailey and co-workers,[11, 12] by the use of injection techniques, were able to demonstrate that it is anatomically possible for a perfusate introduced into the coronary sinus to reach the capillary bed by flowing in retrograde fashion. However, it remains to be demonstrated that this retrograde perfusion of the myocardial capillary bed actually occurs in the sinus-arterialized heart of the living animal.

We have been able to confirm the observation made by Hahn and Beck[5] in demonstrating a significant protection to the heart against ventricular fibrillation following major coronary artery ligation in dogs having a chronically arterialized coronary sinus.[13] Our recent interest lies in the physiologic explanation of this protection. Accordingly, in a series of experiments, we have attempted to elucidate the following points with respect to chronic arterialization of the coronary sinus in dogs: (1) Whether or not the myocardial capillary

bed can actually be perfused in retrograde fashion, (2) if retrograde blood flow does occur, whether or not this blood traverses the capillary bed with extraction of oxygen, and (3) whether or not there is an increase in the inter-arterial coronary collateral flow. In order to obtain this information, we felt that it was necessary to obtain control data first. The following information was therefore secured in the normal dogs: (1) Coronary sinus pressure and oxygen content of coronary sinus blood, (2) quantitative determination of retrograde blood flow from the circumflex coronary artery following its ligation, from a point distal to the ligature, and (3) determination of oxygen content and saturation upon simultaneously drawn blood samples from the pulmonary artery, aorta, and the distal segment of the transected circumflex coronary artery. Inasmuch as it might be questioned whether the production of pericardial adhesions following any cardiac procedure might alter these findings, we felt it essential to repeat these experiments in dogs having previously had nondescript cardiac operations. When possible, dogs were used in which the coronary sinus had been unsuccessfully arterialized, with subsequent thrombosis of the graft.

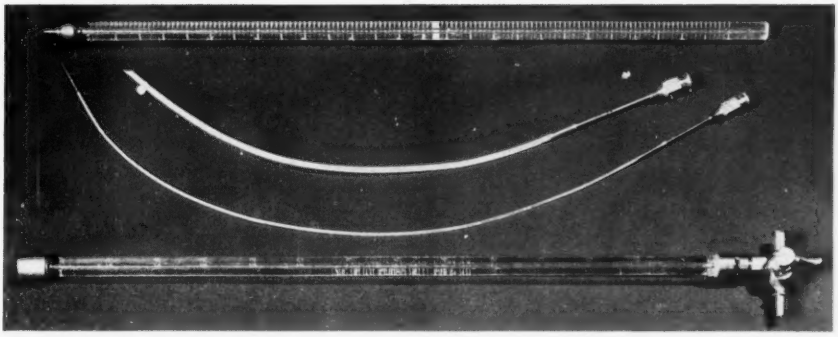

Fig. 1.—The needle-tipped polythene catheter (*center, bottom*) is inserted into the coronary sinus and attached to the water manometer. This permits the securing of accurate pressure readings. The obliquely cut polythene catheter (*center, top*) is used to cannulate the distal segment of the ligated left circumflex artery. The cannula fits snugly within the lumen of the vessel and prevents leakage of blood around it, thus safeguarding accurate retrograde arterial flow studies.

METHODS

Group A, consisting of ten mongrel dogs whose weights varied between 11 and 17 kilograms, constituted the normal control. They were anesthetized with Halatal,* and under artificial respiration the left thorax was opened through the fourth intercostal space. The pericardium was slit, and with the aid of a needle-tipped polythene catheter, the coronary sinus was cannulated. The cannula was connected to a water manometer (Fig. 1) and the sinus pressure was obtained (Fig. 2). A specimen of coronary sinus blood was drawn. The circumflex branch of the left coronary artery was isolated and ligated at its origin. An incision was made in the artery just distal to the ligature, through

*A preparation of pentobarbital sodium.

which the distal segment of the artery was cannulated. Simultaneous blood samples were taken from the pulmonary artery, the aorta, and the distal segment of the ligated circumflex artery. The minute volume of retrograde blood flow from the cut end of the distal portion of the circumflex artery was secured (Fig. 3). The oxygen content of all blood samples was determined. Oxygen capacity and saturation were determined in the aortic sample obtained from these normal control animals.

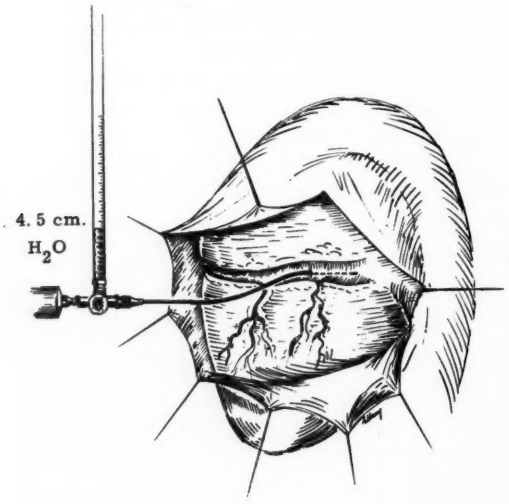

Fig. 2.

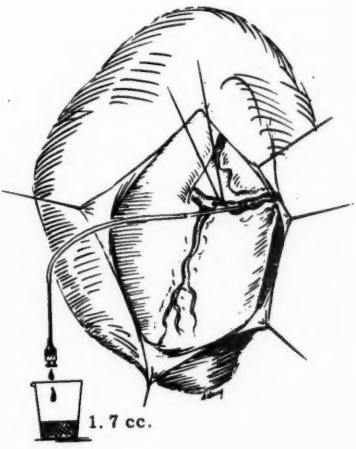

Fig. 3.

Fig. 2.—A needle-tipped polythene catheter is attached to a water manometer, and inserted into the coronary sinus. The recording of the mean coronary sinus pressure is doubly checked, since the fluid level in the manometer is permitted first to fall, and then rise to its end point. A blood specimen is then obtained for the determination of oxygen content.

Fig. 3.—The circumflex branch of the left coronary artery is dissected to its origin, where it is ligated. An incision is made in the artery distal to the ligature, and a polythene cannula is inserted. Retrograde blood flow from this distal cannulated segment of circumflex coronary artery is recorded, and blood samples are secured for the determination of oxygen content.

Group B consists of ten mongrel dogs of similar weight on which nondescript cardiac surgery had been previously performed. In four of these dogs, a previous unsuccessful attempt to arterialize the coronary sinus had been made, but the grafts were demonstrated to be thrombosed and nonfunctioning. The remaining dogs had had the right atrium opened for periods of six to twelve minutes during hypothermia experiments performed one to three months previously. In addition, in some of these, interatrial septal defects had been created and closed. In five of the dogs, the anterior descending branch of the left coronary artery was ligated at its origin, and in the remaining five, the circumflex branch of the left coronary artery was ligated at its origin. The distal segment of the ligated vessel was then cannulated.

RESULTS

1. *Retrograde Blood Flow From the Distal Segment of the Ligated Circumflex Branch of the Left Coronary Artery.—*

Group A: The results in the dogs in this group are shown in Table I. The retrograde bleeding ranged from 0.4 c.c. to 5.0 c.c., with an average of 1.7 c.c. per minute.

Group B: The results are shown in Table I. Retrograde bleeding from the distal segment of the ligated circumflex branch of the left coronary artery ranged from 0.5 c.c. to 6.0 c.c., with an average of 1.9 c.c. per minute. Retrograde bleeding from the left anterior descending artery ranged from 1.8 c.c. to 11.0 c.c. per minute, with an average of 5.1 c.c. per minute. It should be noted that in two dogs (Table I, Group B, 9, 10) there was an unusually large amount of retrograde bleeding. In both animals, a large accessory anterior descending branch was observed to arise from the circumflex artery, and to course downward over the left ventricle parallel to the ligated left anterior descending artery. This accessory artery may have accounted for the excessive retrograde bleeding by providing additional intercoronary communications.

TABLE I. RETROGRADE BLOOD FLOW FROM THE DISTAL CUT END OF THE LIGATED CIRCUMFLEX BRANCH OF THE LEFT CORONARY ARTERY (CUBIC CENTIMETERS PER MINUTE)

GROUP A: NORMAL DOGS		GROUP B: DOGS WITH PREVIOUS CARDIAC SURGERY	
1.	2.2	1.	1.1
2.	1.4	2.	1.2
3.	4.4	3.	1.2
4.	0.4	4.	6.0
5.	0.4	5.	0.5
6.	0.5	Average	1.9
7.	0.6		
8.	1.1	*Left Anterior Descending Artery*	
9.	1.0		
10.	5.0	6.	1.4
Average	1.7	7.	1.8
		8.	1.5
		9.	11.0
		10.	6.2
		Average	5.1

2. *Oxygen Content of the Retrograde Blood Samples Obtained From the Distal Segment of the Ligated Left Circumflex Artery.—*

Group A: The oxygen content of the aortic, pulmonic, and retrograde coronary artery blood varied from dog to dog. However, the retrograde coronary arterial blood was always highly oxygenated, and its oxygen content invariably approximated that of simultaneously drawn aortic blood. The oxygen content of the blood obtained from the distal cut end of the ligated circumflex artery ranged from 8.1 to 18.0 volumes per cent with an average of 14.5 volumes per cent. Concomitant samples of aortic blood ranged from 7.2 to 18.8 volumes per cent with an average of 15.3 volumes per cent (see Table II).

Group B: The findings in this group were almost identical to those of Group A. The oxygen content of the retrograde circumflex blood samples varied from dog to dog. The blood was invariably highly oxygenated, and always approximated the oxygen content of aortic blood. These values ranged from 11.4 to 24.0 volumes per cent with an average of 16.6 volumes per cent. Concomitant samples of aortic blood ranged from 10.7 to 23.4 volumes per cent with an average of 16.5 volumes per cent (see Table II).

TABLE II. OXYGEN CONTENT OF THE RETROGRADE BLOOD FROM THE DISTAL CUT END OF THE LIGATED CIRCUMFLEX ARTERY

	NORMAL DOGS					DOGS WITH PREVIOUS CARDIAC SURGERY			
	O_2 CONT. RETROGR. COR. ART. (VOL. %)	O_2 CONT. AORTIC BLOOD (VOL. %)	O_2 CAP. AORTIC BLOOD (VOL. %)	% SAT'N AORTIC BLOOD	O_2 CONT. PUL. ART. (VOL. %)		O_2 CONT. RETROGR. COR. ART. (VOL. %)	O_2 CONT. AORTIC BLOOD (VOL. %)	O_2 CONT. PUL. ART. (VOL. %)
1.	17.3	18.8			10.2	1.	17.3	16.4	12.2
2.	15.1	15.8	16.8	94.8	12.6	2.	24.0	23.4	20.6
3.	17.5	18.2	18.2	100.0	12.2	3.	11.4	10.7	4.6
4.	14.7	16.2	16.7	100.0	10.9	4.	17.8	18.0	11.9
5.	15.8	16.9	17.8	94.9	14.5	5.	14.0	14.5	10.6
6.	18.0	17.5	17.8	98.3	11.5		Average		
7.	15.4	16.3	17.1	95.3	11.6		16.6	16.5	10.9
8.	8.1	7.2	12.2	59.8	4.8	*Left Anterior Descending Artery*			
9.	11.6	11.5	17.4	66.0	8.6	6.	18.3	17.2	10.1
10.	11.9	14.7	19.5	75.4	9.5	7.	13.6	15.0	10.0
	Average					8.	14.9	14.9	10.1
	14.5	15.3			10.6	9.	18.0	17.1	13.0
						10.	17.2	17.5	10.5
							Average		
							16.4	16.3	10.5

3. *Mean Coronary Sinus Pressure and Oxygen Content.*—

Group A: The mean coronary sinus pressure ranged from 2 to 12 cm. of water with an average of 6.1 cm. of water. The coronary sinus blood oxygen content ranged from 1.0 to 9.2 volumes per cent with an average of 4.5 volumes per cent (see Table III).

TABLE III. MEAN CORONARY SINUS PRESSURE AND OXYGEN CONTENT OF CORONARY SINUS BLOOD

	NORMAL DOGS		DOGS WITH PREVIOUS CARDIAC SURGERY	
	COR. SINUS PRESSURE (CM. H_2O)	O_2 CONT. COR. SINUS (VOL. %)	COR. SINUS PRESSURE (CM. H_2O)	O_2 CONT. COR. SINUS (VOL. %)
1.	12.0	9.2	5.4	
2.	4.0	6.3	6.0	
3.	2.0	6.7	(23.0)	
4.	5.0	4.4	12.0	
5.	8.6	2.9	6.4	4.8
6.	8.4	7.4	4.0	4.3
7.	4.6	3.6	4.0	
8.	4.0	1.0	7.0	
9.	7.0	2.4	7.2	
10.	5.0	1.3	10.0	
Average	6.1	4.5	6.7	

Group B: The mean coronary sinus pressure ranged from 4.0 to 12 cm. of water with an average of 6.7 cm. of water. Dog 3, Group B, had a mean coronary sinus pressure of 23 cm. of water. It is not included in this average since it was in severe right heart failure, and was included only to determine the effect of elevated coronary sinus pressure without sinus occlusion upon retrograde coronary artery bleeding. Coronary sinus blood oxygen content was determined in two dogs, and was 4.8 and 4.3 volumes per cent, respectively (see Table III).

4. *Mortality of Circumflex Coronary Artery Ligation.*—

Group A: Ligation of the circumflex branch of the left coronary artery at its origin in a series of ten normal dogs was associated with a 100 per cent mortality. Nine of the ten dogs died of ventricular fibrillation within the first thirty minutes following circumflex arterial ligation. The majority of them developed ventricular fibrillation within the first few minutes following ligation. One dog died thirty-six hours postoperatively.

Group B: In the group of dogs (1 to 5) in which nondescript surgery had been performed previously, after the circumflex branch was ligated, four died of ventricular fibrillation within twenty minutes, and one survived for forty days. In the group (6 to 10) in which the left anterior descending artery was ligated, one died of cardiac arrest in seventeen minutes; one died of ventricular fibrillation within twenty minutes; one died in forty-eight hours; two survived until sacrificed. In both survivors (Dogs 9, 10, Group B, Table I), a large accessory anterior descending artery arising from the circumflex was demonstrated, and in both, the value of the retrograde blood flow from the distal cut end of the vessel was excessively high.

DISCUSSION

The results obtained in this experiment emphasize several points. Most important is the fact that the retrograde blood obtained from the distal segment of the ligated circumflex coronary artery in the normal dog is highly oxygenated, and arterial in nature. In all instances, its oxygen content approximates the values of blood specimens simultaneously obtained from the aorta. Since the blood flow from the distal segment of the ligated artery is continuous and of high oxygen content, its source must be from neighboring coronary arteries. This retrograde blood flow from the distal segment of the ligated coronary artery in dogs undoubtedly arises from small interarterial communications, and represents interarterial coronary collateral flow. Therefore, quantitating the retrograde coronary arterial flow provides an accurate method of measuring and comparing interarterial coronary collateral flow. This method of quantitating interarterial coronary collateral flow is useful in evaluating the effect of cardiac procedures designed to increase the collateral circulation of the heart in occlusive coronary artery disease.

In those dogs which had had previous nondescript cardiac surgery, with formation of pericardial adhesions, the findings were similar to those in the normal dogs. The retrograde blood flow from the distal segment of the ligated circumflex artery was arterial in nature and small in quantity, measuring approximately 1.9 c.c. per minute. Thus, nondescript cardiac surgery, per se, does not noticeably stimulate an increase in the interarterial coronary collateral flow.

The mean coronary sinus pressure under the conditions of our experiment averaged 6.1 cm. of water, and the oxygen content of coronary sinus blood averaged 4.5 volumes per cent. This was similar to the findings of Gregg and associates[15-17] in their studies.

In Dog 3, Group B, the mean coronary sinus pressure was elevated because of severe right heart failure. The retrograde circumflex coronary arterial flow

was similar to that in the normal dogs. According to Eckstein and co-workers,[10] progressive elevation in coronary sinus pressure produces proportionate increases in retrograde coronary flow.

The mortality in these experiments, from ligation of the circumflex branch of the left coronary artery, was 100 per cent. Ligation of the left anterior descending branch in our series of dogs produced a 60 per cent mortality. This was similar to the mortality following left anterior descending coronary ligation in normal dogs reported by Hahn and Beck.[5] The incidence of the occurrence of an accessory left anterior descending coronary artery in our series was 40 per cent, which corresponds with the survival rate. It is possible that the survival from ligation of the anterior descending branch of the coronary artery in our experiments, and in those of Hahn and associates, are attributable to the fortuitous presence of such accessory branches.

SUMMARY

1. An accurate method of measuring interarterial coronary collateral flow is described.

2. Retrograde coronary arterial blood flow and the oxygen content of blood obtained from the distal segment of the circumflex coronary artery have been determined in a series of normal dogs. Similar studies were performed in a second series of dogs in which nondescript cardiac surgery had been previously performed.

3. In both groups, the retrograde bleeding was of small quantity and arterial in nature, always approximating the oxygen content of aortic blood. This retrograde blood flow is believed to be due to interarterial coronary collateral circulation.

4. The mortality due to ligation of the circumflex branch of the left coronary artery in ten normal dogs was 100 per cent. The mortality due to ligation of the left anterior descending artery was 60 per cent. The higher survival rate in the latter group may be due to the high incidence in this small series of an accessory left anterior descending coronary artery arising from the circumflex branch of the left coronary artery.

The authors wish to express their appreciation to Miss Betty Underwood and Miss Ruth Freeman for invaluable technical assistance.

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PROTECTION OF THE HEART BY ARTERIALIZATION OF THE CORONARY SINUS

II. Coronary Flow in Dogs With Aorticocoronary Sinus Anastomosis

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THIS is a continuation of previous studies undertaken to investigate and evaluate the benefits to the heart in occlusive coronary artery disease afforded by arterialization of the coronary sinus. The original conception for the revascularization of the myocardium was first described by Pratt[1] in 1898, who perfused the coronary sinus of a freshly extirpated cat's heart with defibrinated arterialized blood, and was able to maintain regular coordinated contractions for one and one-half hours. Gross and associates[2] demonstrated that ligation of the coronary sinus in dogs elevated the coronary sinus pressure. This procedure was associated with a very low operative mortality. They demonstrated a reduction in the mortality and size of infarcts following major coronary artery occlusion in sinus-ligated dogs. Roberts and co-workers[3] demonstrated in dogs that following the creation of an arteriovenous anastomosis between a systemic artery and the coronary sinus, forceful cardiac action was maintained for as long as twenty-six hours following coronary artery ligation.

Stenstrom,[4] in dogs with sinus arterialization, demonstrated a reduction in the number of infarcts and incidence of ventricular fibrillation following coronary artery ligation. Hahn and Beck[7] have demonstrated a marked benefit afforded by coronary sinus arterialization in major coronary artery ligation in dogs.

The procedure developed by Beck and his associates is aimed at the revascularization of the heart in occlusive coronary artery disease. It utilizes the placement of a free vein graft between the aorta and the coronary sinus, followed by the subsequent partial ligation of the coronary sinus immediately proximal to its entrance into the right atrium. This second stage of the procedure prevents the complete egress of the blood into the right atrium, and forces a portion of the aortic blood into the venous tree.

Arterialization of the coronary sinus has been demonstrated to protect the heart from ventricular fibrillation following coronary artery ligation.[7, 10, 11] It also reduces the number and size of myocardial infarctions in those dogs surviving coronary artery ligation, as well as favorably altering the survival rate in these experiments. Bailey and co-workers[13] have demonstrated that,

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anatomically, fluid introduced into the coronary sinus may reach the capillary bed in retrograde fashion. However, it remains to be proved that following coronary artery ligation the protection afforded by coronary sinus arterialization is on the basis of retrograde myocardial capillary flow, with improvement of myocardial oxygenation.

Inasmuch as the arteriovenous tree is continuous both directly and via the capillary bed, it seemed reasonable that a portion of the arterial blood introduced into the coronary sinus following sinus arterialization might be recovered in retrograde fashion from the distal end of a transected coronary artery. In order to determine the effects of arterialization of the coronary sinus on this retrograde coronary arterial blood flow, it was first necessary to establish normal control values for the quantity and oxygen content of this retrograde coronary arterial blood. Moreover, we have repeated these experiments in a series of control animals having had previous nondescript cardiac surgery to analyze the effect of previous cardiac surgery and pericardial adhesions on the retrograde coronary arterial blood flow and oxygen content. When possible, animals having had previous unsuccessful aorticocoronary sinus anastomoses, with thrombosis of the graft, were utilized.

At this point, having established control values in our series of normal dogs and those with previous nondescript cardiac surgery, we repeated these experiments in dogs protected by aorticocoronary sinus anastomoses with the graft open and closed.

METHOD

Ten mongrel dogs were first prepared with arterialization of the coronary sinus, using the two-stage operation developed by Beck, and modified by us. At a first stage, through a left-sided approach, an external jugular vein graft was placed between the aorta and the coronary sinus. Three to four weeks later, through a right-sided approach, the coronary sinus was partially ligated over a probe to 2 to 3 mm. Four to eight weeks following the second stage of the procedure, physiologic data were obtained. The animals were anesthetized with Halatal, after which artificial respiration was maintained using intermittent positive pressure through an endotracheal tube. The left chest was entered through the fourth intercostal space, and dense pleural and pericardial adhesions were divided. The vein graft was dissected, and examined for patency. Only dogs with patent grafts were used. An umbilical tape was passed around the graft to facilitate its easy occlusion at the appropriate time during the procedure. A needle-tipped polythene catheter connected to a water manometer was inserted into the coronary sinus, after which coronary sinus pressure and blood samples for oxygen content were obtained (Fig. 1). The circumflex branch of the left coronary artery was carefully dissected to its origin, and ligated. The artery was cannulated distal to the ligature, and a sample of retrograde blood was collected (Fig. 2). Simultaneous blood samples were taken from the aorta and the pulmonary artery. The minute volume of retrograde blood flow was then recorded. At this point the graft was occluded, and the experiment was repeated (Figs. 3 and 4). This was the method used for

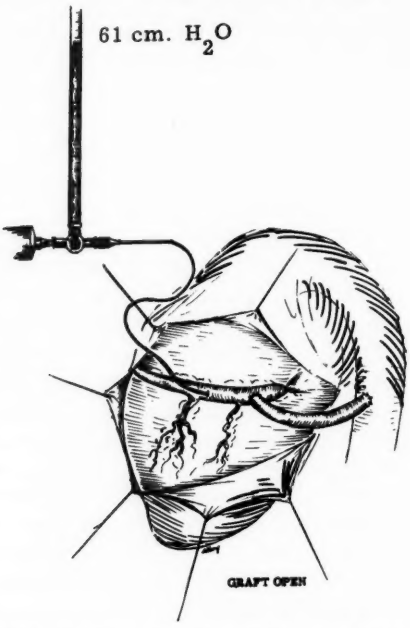

Fig. 1.—A needle-tipped polythene catheter attached to a water manometer is inserted into the coronary sinus proximal to the site of partial ligation. Coronary sinus pressure is frequently above the recordable level of the manometer when its base is maintained level with the sinus. The recorded readings are corrected by raising the base of the manometer above the coronary sinus level, measuring the distance, and adding it to the manometric reading.

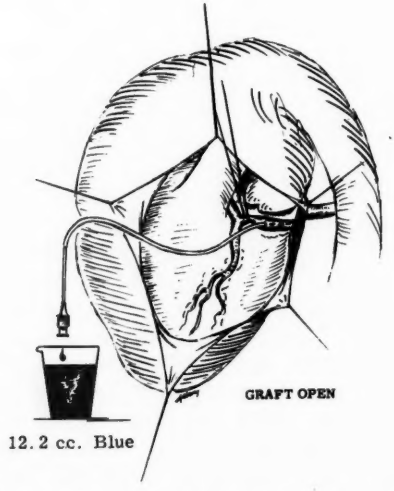

Fig. 2.—The circumflex branch of the left coronary artery is carefully dissected to its origin, where it is ligated. An incision is made in the artery distal to the ligature, and a polythene catheter is inserted. Retrograde blood flow from this distal cannulated segment of circumflex coronary artery is recorded, and blood samples are secured for the determination of oxygen content.

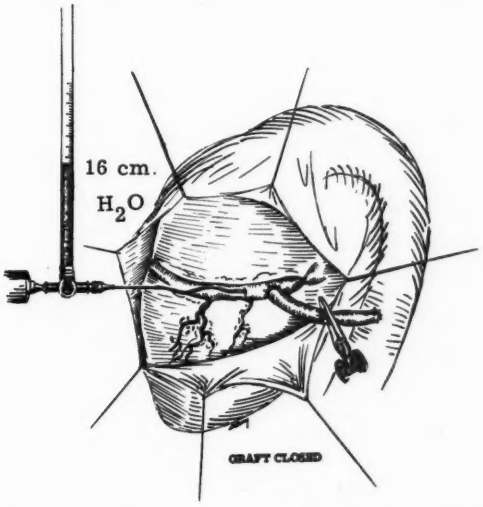

Fig. 3.—A needle-tipped polythene catheter was previously inserted into the coronary sinus proximal to the site of partial ligation. This catheter is attached to a water manometer. The aorta to coronary sinus vein graft is occluded, and the fall in coronary sinus pressure is recorded. Following the occlusion of the graft, a period of two minutes is permitted to elapse before a blood specimen is secured for determination of blood oxygen content.

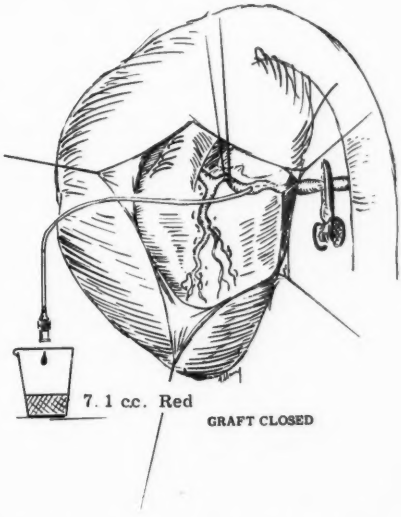

Fig. 4.—The left circumflex artery was previously ligated at its origin, and the distal segment was cannulated with a polythene catheter. Following the occlusion of the aorta to coronary sinus vein graft, the distal end of the transected coronary artery is permitted to bleed freely through the cannula for a period of two minutes, during which time minute blood flow is recorded. At the end of this interval of time, a blood sample is secured for the determination of blood oxygen content.

TABLE I. RETROGRADE BLOOD FLOW AND OXYGEN CONTENT OF BLOOD FROM THE DISTAL END OF THE TRANSECTED LEFT CIRCUMFLEX CORONARY ARTERY IN SINUS-ARTERIALIZED DOGS WITH THE GRAFT OPEN AND CLOSED

	GRAFT OPEN				GRAFT CLOSED							
	RETR. COR. ART.		CORONARY SINUS		RETR. COR. ART.		CORONARY SINUS		AORTIC BLOOD			PUL. ART.
DOG NO.	FLOW (C.C./MIN.)	O_2 CONT. (VOL. %)	PRESS. (CM. H_2O)	O_2 CONT. (VOL. %)	FLOW (C.C./MIN.)	O_2 CONT. (VOL. %)	PRESS. (CM. H_2O)	O_2 CONT. (VOL. %)	CONT. (VOL. %)	CAP. (VOL. %)	SAT'N (%)	O_2 CONT. (VOL. %)
					Blood Drawn Immediately Following Occlusion of Graft							
1.	9.6	9.0	45						15.2			10.3
2.	9.0	1.9	80	17.2	7	5.3			18.8	20.6	91.3	11.4
3.	11.0	11.8	60	12.5					19.2	20.7	92.8	15.9
4.	12.0	6.1	36	12.4	10	7.7			14.0	15.8	99.6	11.1
5.	12.0	1.5	60	9.7	7	4.6	20		12.1	13.0	93.1	10.2
					Blood Drawn Two Minutes Following Occlusion of Graft							
6.	10.0	4.8	71	15.5	2	12.5	9		17.7	18.4	96.2	14.3
7.	18.0	4.0	64	11.9	8	13.2	19	7.1	14.3	14.0	100.0	9.2
8.	10.0	15.1	59	17.9	8	18.5	18		22.5	22.5	100.0	19.1
9.	14.0	4.8	68	13.2	7	15.3	16	1.7	17.2	20.1	85.6	7.6
10.	16.0	3.9	70	15.6	8	17.9	14	1.9	17.8	19.8	89.9	11.6
						Average						
	12.2	6.3	61	14.0	7.1	15.5	16	3.6	17.0			12.1

the study of the first five dogs. During the experiment on the sixth animal, following the occlusion of the vein graft, the cannula was inadvertently dislodged from its position in the distal circumflex artery. It was noted that the retrograde arterial blood, which was blue prior to the clamping of the graft, remained so immediately thereafter. The graft remained occluded during the period of time which elapsed while the artery was recannulated. At the end of this period of time, it was observed that the retrograde blood had changed from blue to red. Consequently, in Dogs 6 to 10, following the occlusion of the graft, the distal circumflex artery was permitted to bleed freely for two minutes prior to the collection of blood samples for oxygen content.

RESULTS

1. *Retrograde Blood Flow From the Distal Cut End of the Ligated Circumflex Branch of the Left Coronary Artery.*—The results are shown in Table I. With the graft open, retrograde blood flow ranged from 9.0 to 18.0 c.c. per minute, with an average of 12.2 c.c. per minute.

Following the occlusion of the aorticocoronary sinus vein graft, the retrograde circumflex artery blood flow fell significantly in each dog. These values ranged from 2 to 10 c.c. per minute, with an average of 7.1 c.c. per minute.

2. *Oxygen Content of the Retrograde Blood Collected From the Distal End of the Transected Circumflex Artery.*—The results are shown in Table I. Although the oxygen content of this blood varied widely from dog to dog, the character of this blood remained invariable. The values for the oxygen content of this retrograde blood ranged from 1.5 to 15.1 volumes per cent, with an average of 6.3 volumes per cent. Concomitant samples of aortic blood ranged from 12.1 to 22.5 volumes per cent, with an average of 17.1 volumes per cent.

Despite the wide range of values of these blood samples, the retrograde circumflex arterial blood was constantly venous in nature and its oxygen content invariably much lower than pulmonary arterial blood. Moreover, the oxygen content of this retrograde circumflex arterial blood was within the limit of the values usually seen in normal coronary sinus blood. In fact, the average oxygen content of the retrograde circumflex arterial blood was 6.3 volumes per cent, which compares favorably with the average value for the oxygen content of normal coronary sinus blood, which was 4.5 volumes per cent.

Following the clamping of the vein graft, the values obtained for the oxygen content of the retrograde circumflex arterial blood divide themselves into two groups. The first group consists of Dogs 1 to 5, in which the blood was collected immediately following the clamping of the graft. The second group consists of Dogs 6 to 10, in which the ligated circumflex artery was permitted to bleed freely in retrograde fashion for a period of two minutes prior to the collection of the blood samples. Inasmuch as these two sets of values are significantly different, we are reporting them separately. In Dogs 1 to 5, the oxygen content of the retrograde circumflex blood ranged from 4.6 to 7.7 volumes per cent with an average of 5.8 volumes per cent. These values are almost identical with the values of retrograde blood oxygen content with the graft open. However, upon careful analysis of the data from the individual dogs, in each case there is a

significant rise in the oxygen content of the retrograde circumflex arterial blood immediately after the graft is occluded. In Dog 2, retrograde circumflex blood oxygen content was 1.9 volumes per cent with the graft open, and rose to 5.3 volumes per cent immediately after it was clamped; in Dog 4, the value rose from 6.1 to 7.7 volumes per cent; in Dog 5, from 1.5 to 4.6 volumes per cent. We believe it probable that were the artery permitted to bleed freely in retrograde fashion for two minutes, the values obtained in Dogs 1 to 5 would have risen further toward the content of arterial blood, and would have more closely resembled the findings in Dogs 6 to 10.

The second group of animals (6 to 10) consists of those in which the distal cut end of the ligated circumflex artery was permitted to bleed freely in retrograde fashion for a period of two minutes prior to the collection of the blood samples. In this group, the retrograde circumflex arterial blood oxygen content ranged from 12.5 to 18.5 volumes per cent, with an average of 15.5 volumes per cent. Despite the wide range of values, the retrograde circumflex arterial blood oxygen content compares favorably with that of aortic blood. Concomitant samples of aortic blood in Dogs 6 to 10 ranged from 14.3 to 22.5 volumes per cent, with an average of 17.9 volumes per cent. Of significance is the fact that the blood oxygen content of the retrograde circumflex arterial blood rose significantly from an average of 6.3 volumes per cent with the graft open to an average of 15.5 volumes per cent following occlusion of the graft, and changed in appearance from dark venous to bright arterial blood.

3. *Mean Coronary Sinus Pressure and Blood Oxygen Content.*—With the graft open, mean coronary sinus pressure ranged from 36 to 80 cm. of water, with an average of 61 cm. of water. The oxygen content of the arterialized coronary sinus blood ranged from 9.7 to 17.9 volumes per cent with an average of 14.0 volumes per cent. It may be seen in Fig. 5 that there is no direct relationship between the retrograde circumflex arterial blood flow and coronary sinus pressure.

Following occlusion of the aorticocoronary sinus graft, sinus pressure fell significantly toward, but not quite to normal. The pressures ranged from 9 to 20 cm. of water with an average of 16 cm. of water.

In a few dogs, coronary sinus blood samples were drawn two minutes after the graft was clamped. The values for the oxygen content of these samples ranged from 1.7 to 7.1 volumes per cent, with an average of 3.6 volumes per cent. Thus, in these animals, after the graft was clamped the coronary sinus blood oxygen content fell from values within arterial range to values within normal sinus range.

DISCUSSION

The most striking finding of the experiment revolves around the marked change in the amount and nature of the retrograde blood from the distal segment of the ligated and transected circumflex coronary artery in sinus-arterialized dogs as opposed to those in normal dogs. In our previous communication,[15] retrograde circumflex arterial blood flow in the normal animal ranged from 0.4 to 5.0 c.c. per minute, and averaged 1.7 c.c. per minute. The oxygen content

of this blood ranged from 8.1 to 18.0 volumes per cent with an average of 14.5 volumes per cent. Thus, in the normal animal, retrograde circumflex arterial bleeding from a point distal to the ligature is small in quantity, and is invariably arterial in nature. The findings were similar in dogs having had nondescript cardiac procedures. The arterial nature of this retrograde bleeding from the distal cut end of the ligated circumflex artery signifies that the origin of this blood is arterial. It undoubtedly originates in the nonoccluded coronary

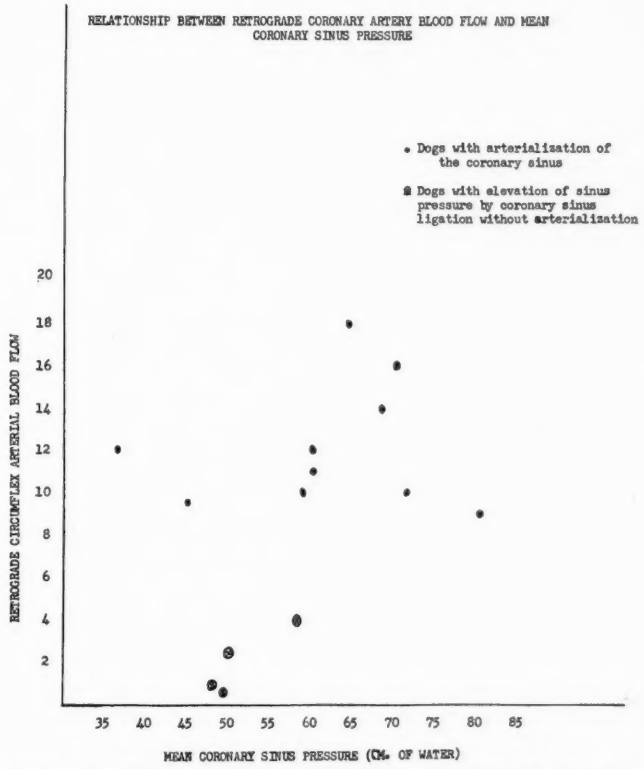
RELATIONSHIP BETWEEN RETROGRADE CORONARY ARTERY BLOOD FLOW AND MEAN CORONARY SINUS PRESSURE
• Dogs with arterialization of the coronary sinus
Dogs with elevation of sinus pressure by coronary sinus ligation without arterialization
RETROGRADE CIRCUMFLEX ARTERIAL BLOOD FLOW
20
18
16
14
12
10
8
6
4
2
35 40 45 50 55 60 65 70 75 80 85
MEAN CORONARY SINUS PRESSURE (CM. OF WATER)

Fig. 5.—Retrograde circumflex arterial blood flow is plotted against the mean coronary sinus pressure. The scatter pattern of the graft does not suggest any direct relationship of the two in this series of experiments.

arteries, and reaches the distal ligated coronary artery through small interarterial anastomoses. Thus, measurement of the retrograde coronary arterial blood flow from the distal end of the ligated and transected coronary artery is a measure of the existing intercoronary arterial anastomotic flow. This was scant in the normal dog, and was unchanged in those dogs having had previous nondescript cardiac operations.

These findings were strikingly altered following arterialization of the coronary sinus for four to eight weeks. Following coronary sinus arterializa-

tion, the quantity of retrograde circumflex arterial blood flow increased markedly from a normal average of 1.7 c.c. to 12.2 c.c. per minute. Arterialization of the coronary sinus also precipitated a change in the oxygen content of this retrograde circumflex arterial blood from a normal average content of 14.5 volumes per cent to 6.3 volumes per cent. These findings unmistakably attest to the increase in the retrograde circumflex arterial flow following aorticocoronary sinus anastomosis. This increase in the retrograde coronary arterial blood flow was similarly noted by Hahn and associates,[7-9] who found that in dogs with coronary sinus arterialization, but with the graft occluded, retrograde coronary arterial bleeding was red and of small quantity. With the graft open, retrograde bleeding increased in quantity, and the blood was blue. Eckstein and co-workers,[10, 11] in experiments on the acutely arterialized coronary sinus, have demonstrated a similar increase in the retrograde flow from the distal cut end of the circumflex artery. In their experiments, the acute arterialization of the coronary sinus was effected by using a glass cannula to connect a systemic artery to the coronary sinus. They injected P32 into the coronary sinus, and in thirteen seconds recovered it from the distal ligated coronary artery.

It should be noted that with the graft open, and the coronary sinus arterialized, the oxygen content of coronary sinus blood averaged 14.0 volumes per cent, and the pressure averaged 61 cm. of water. Thus, highly oxygenated blood was delivered to the coronary sinus under high pressure. Of extreme significance is the fact that blood collected from the distal cut end of the ligated circumflex artery in the sinus-arterialized dogs of four to eight weeks' duration is venous in character, its oxygen content falling within the range of normal coronary sinus blood. This is in contrast to the normal dog, in which it is arterial. We have demonstrated that nonspecific cardiac procedures per se do not alter the quantity or the nature of the retrograde blood found in the normal animal. The change in character and quantity of the retrograde blood follows coronary sinus arterialization, and is accomplished by elevation of the mean coronary sinus pressure to arterial levels (61 to 70 cm. of water), and by the introduction of highly oxygenated blood into the coronary sinus.

Under these circumstances, the blood recovered from the distal cut end of the circumflex artery is large in quantity and of low oxygen content. The low oxygen content of this blood is in the magnitude of normal coronary sinus blood oxygen content, and demonstrates that it has traversed the capillary bed with extraction of oxygen by the myocardium.

In the normal animal, the pressure gradient falls from the arterial to the venous end of the capillary bed. This procedure elevates coronary sinus pressure, thereby completely altering the normal pressure gradient, and favoring the retrograde flow of oxygenated blood. Following occlusion of the graft, the pressure gradients again favor the normal direction of flow, and an almost normal situation is obtained. Coronary sinus pressure falls toward, but not quite to normal, and retrograde coronary artery bleeding again becomes arterial in nature, both simulating the normal dog (Fig. 6). It may be significant that coronary sinus pressure does not fall to normal. Gregg and associates[18-23] have

demonstrated that following coronary sinus ligation, retrograde bleeding from the distal cut end of the descending ramus of the left coronary artery increased in quantity, and became venous in nature. They noted that following concomitant circumflex artery ligation, the back flow from the left anterior descending artery decreased appreciably. It may be inferred that the residual elevation of coronary sinus pressure may be a factor in the increased retrograde circumflex arterial blood flow following occlusion of the graft. Eckstein and coworkers[11] have demonstrated a proportionate relationship between the retrograde arterial bleeding and coronary sinus pressure in their acute experiments.

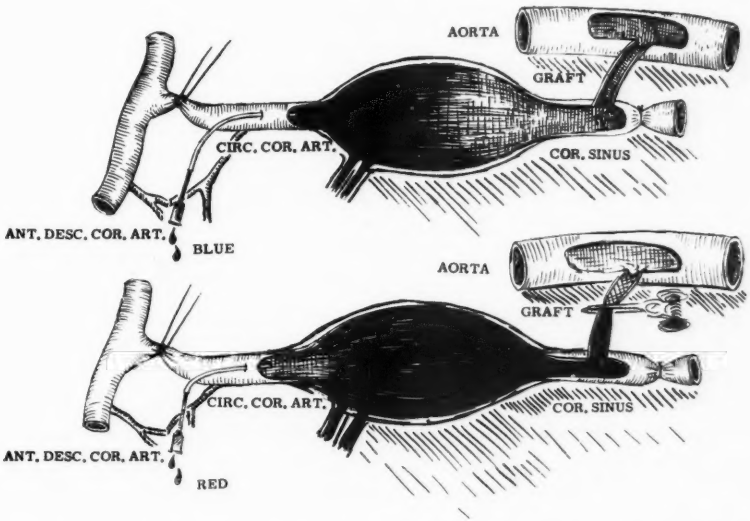

Fig. 6.—*A*, Aorta to coronary sinus anastomosis produces an elevation of coronary sinus pressure, and introduces highly oxygenated blood into the coronary sinus. This alters the normal pressure gradient, and favors the retrograde flow of a portion of this oxygenated blood.

B, Following occlusion of the graft, the pressure gradient again favors the normal direction of flow, and an almost normal situation is obtained. Coronary sinus pressure falls toward normal, and retrograde blood from the distal segment of the coronary artery again becomes arterial in nature, both simulating the normal dog.

We did not note this relationship in dogs in which the coronary sinus had been arterialized for four to eight weeks (Fig. 5). In addition, in several animals in which the coronary sinus pressure was elevated to 48 to 58 cm. of water by complete sinus occlusion, retrograde circumflex arterial blood flow averaged 2.5 c.c.[17] Although coronary sinus pressure was elevated eight to ten times above normal, the flow from the distal cut end of the circumflex artery was within normal range.

Following occlusion of the graft, the volume of retrograde coronary arterial bleeding from the distal cut end of the circumflex artery falls from 12.2 c.c. per minute with the graft open to 7.1 c.c. per minute. This blood is highly oxygenated and arterial in nature, and undoubtedly represents blood arising

from intercoronary anastomotic flow. Retrograde bleeding in our normal group was 1.7 c.c. per minute as opposed to an average of 7.1 c.c. per minute in this group. This represents a fourfold increase above the expected normal values, and probably represents a significant increase in the intercoronary arterial anastomotic flow.

When retrograde bleeding from the distal cut end of the circumflex artery is permitted in sinus-arterialized dogs with the graft open, these values rise still higher to an average of 12.2 c.c. per minute. This blood is of very low oxygen content, averaging 6.3 volumes per cent. It is therefore unlikely that the retrograde blood flow with the graft open is significantly contributed to by blood arising from intercoronary anastomoses because of the low oxygen content of this blood. It is more probable that this increased retrograde blood flow from the distal cut end of the ligated circumflex artery represents retrograde flow from the coronary sinus. The venous nature of this blood is incontrovertible evidence that it has traversed the myocardial capillary bed, where its oxygen was extracted.

In our experiments on sinus-arterialized dogs, we have observed that following circumflex coronary artery ligation, the heart continued to beat in vigorous, uninterrupted fashion. Whereas we found it imperative to expedite the collection of data in the normal animals before ventricular fibrillation ensued, we soon learned that we could proceed more deliberately in the sinus-arterialized animals, provided the graft was functioning. However, when the graft was occluded, data had to be obtained with dispatch lest the heart fibrillate. In several dogs in which the heart beat well for considerable periods of time following circumflex arterial ligation, ventricular fibrillation ensued within five minutes after the graft was clamped. This observation further emphasized the value of the protection afforded by the aorta to coronary sinus anastomosis. These observations are in accordance with those found by others.

Thus, in our experiments we have demonstrated three salient findings which elucidate the protection to the heart afforded by arterialization of the coronary sinus of four to eight weeks' duration following circumflex artery ligation. (1) The myocardial capillary bed is perfused in retrograde fashion, (2) the myocardium is capable of extracting oxygen from blood supplied to it in retrograde fashion, and (3) there is a significant increase in the intercoronary arterial anastomotic flow.

SUMMARY AND CONCLUSIONS

1. Retrograde blood flow and oxygen content from the distal cut end of the ligated and transected circumflex artery have been determined in sinus-arterialized dogs with the graft open and closed.

2. When the graft was open, coronary sinus pressure was high and coronary sinus blood was of high oxygen content.

3. With the graft open, the backflow from the distal cut end of the circumflex coronary artery was of much greater quantity than normal, was of low

oxygen content, and was venous in nature. This demonstrated a reversal of blood flow through the capillary bed with extraction of oxygen by the myocardium.

4. When the graft was occluded, coronary sinus pressure fell significantly, and the blood oxygen content fell to normal coronary sinus levels. Concomitantly, there was a significant fall in the retrograde circumflex arterial blood flow, which returned to normal arterial blood of high oxygen content. This blood flow was four times greater than the expected normal, and was believed to represent a significant increase in the interarterial coronary collateral anastomoses.

5. The physiologic explanation of the protection to the heart afforded by coronary sinus arterialization is believed to be threefold. (a) The myocardial capillary bed is perfused in retrograde fashion with arterial blood, (b) the myocardium is capable of extracting oxygen from the blood supplied to it in retrograde fashion, and (c) there is a significant increase in the blood flow occurring via intercoronary arterial anastomoses.

The authors wish to express their appreciation to Miss Ruth Freeman and Miss Betty Underwood for their technical assistance.

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THE ARTERIAL OXYGEN AND CARBON DIOXIDE TENSION DURING THE POSTOPERATIVE PERIOD IN CASES OF PULMONARY RESECTIONS AND THORACOPLASTIES

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AFTER a lobectomy has been performed, there is frequently some degree of anoxia for a week. This may be due to poor re-expansion of the remaining lobe or to poor ventilation on the operated side for some time after the operation. In cases of total pneumonectomy, Maier and Cournand (1943) found that the oxygen saturation of the arterial blood returned to a normal level within a few days after operation. In cases of thoracotomies and abdominal operations no anoxia was encountered.

We have observed several instances where five-rib thoracoplasties with apicolysis resulted in more severe postoperative courses than resections in similar cases. This must be due to the combination of compression atelectasis produced by the thoracoplasty and the accumulation of fluid in the apicolysis space plus paradoxical movements of the unstable chest wall. For some time following the operation there is perfusion of nonventilated lung parenchyma due to atelectasis and poor ventilation of the aerated parenchyma on the operated side due to the paradoxical movements and pain.

In this article we wish to report the changes in the arterial oxygen and carbon dioxide tension during the immediate postoperative period in cases of pulmonary resections with and without the combination of thoracoplasties. Moreover, blood samples were taken during voluntary hyperventilation to determine how soon after the operation an effective hyperventilation could be performed.

The measurement of the arterial oxygen tension by the indirect method was found to be more accurate in the detection of small changes in the arterial blood than the saturation method.

METHOD

With an indwelling needle in the femoral artery a blood sample was taken at rest and after the patient had been hyperventilating as much as possible for about twenty-five seconds. Oxygen and carbon dioxide tensions (pO_2 and pCO_2) in the arterial blood were determined according to Riley, Proemmel, and Franke with a slight modification. The saline or saline-Dreft solution has been omitted for washing the capillary. The sodium hydroxide solution is applied for both the capillary washing and the simultaneous CO_2-combining as well, thereby eliminating one source of error. This investigation was performed before operation and at intervals during the postoperative period.

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RESULTS

I. Pneumonectomy.—

Five cases of pneumonectomy with different degrees of pulmonary reserve were investigated.

CASE 1.—In a 56-year-old man a right extrapleural pneumonectomy under a seven-rib thoracoplasty was performed for tuberculous empyema. The left lung was healthy. Maximal breathing capacity was 68 L. per minute. The arterial blood tension measurements in mm. Hg were:

	BEFORE OPERATION		13 DAYS AFTER OPERATION	
	AT REST	HYP. VENT.	AT REST	HYP. VENT.
pO_2	94.3	90.4	78.4	81.6
pCO_2	34.1	41.6	37.1	39.0

CASE 2.—In a 30-year-old man with a left tuberculous empyema and a bronchocutaneous fistula under a ten-rib thoracoplasty, an extrapleural pneumonectomy was performed. Maximum breathing capacity was 80 L. per minute. There had been a spread to the right lung but no active contralateral tuberculosis was demonstrated at the time of operation. After the operation the arterial oxygen tension was low for several days before rising; it did not reach the preoperative level until after more than one week (Fig. 1).

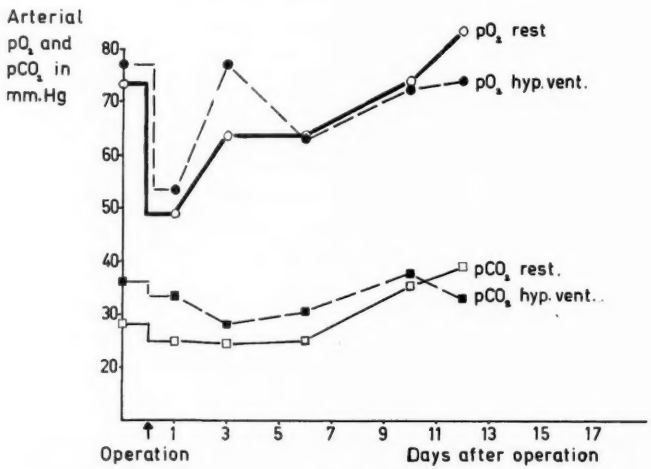


Fig. 1 (Case 2).

CASE 3.—In a 36-year-old man a destroyed left lung was removed. For five years, he had had pneumothorax treatment on the right side for an upper lobe cavity. Maximum breathing capacity was 47 L. per minute. The pressures in the pulmonary artery were normal. Residual volume was 37.4 per cent of the total lung capacity. Not until one week after the operation did the arterial oxygen tension reach the preoperative value (Fig. 2). Resection of the left upper nine ribs was performed one month after the pneumonectomy.

CASE 4.—A 44-year-old man had a right tuberculous empyema with a bronchocutaneous fistula and a left upper lobe cavity. Maximum breathing capacity was 55 L. per minute and his residual volume was 42 per cent. Under a three-rib thoracoplasty a right extrapleural

pneumonectomy with resection of the fourth to the tenth rib was performed. The patient had a low arterial oxygen tension before operation and died eight days after the operation. Autopsy showed a cor pulmonale. The tensions were:

	Before Operation		3 Days After Operation	
	At rest	Hyp. vent.	At rest	Hyp. vent.
pO_2	55.4	57.1	68.3	67.0
pCO_2	42.0	51.7	51.4	48.7

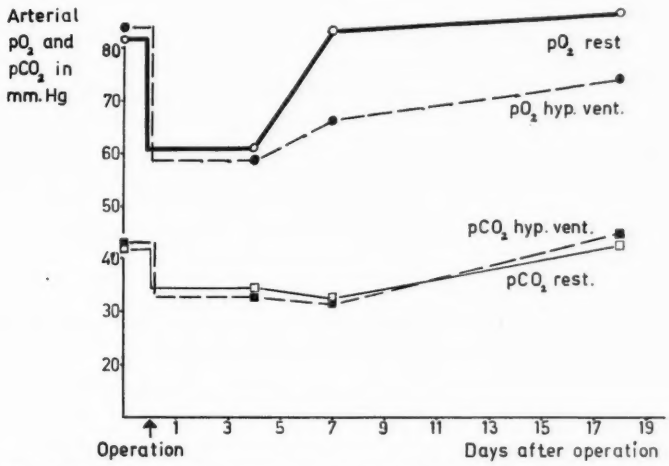


Fig. 2 (Case 3).—Left pneumonectomy performed on patient with a destroyed left lung.

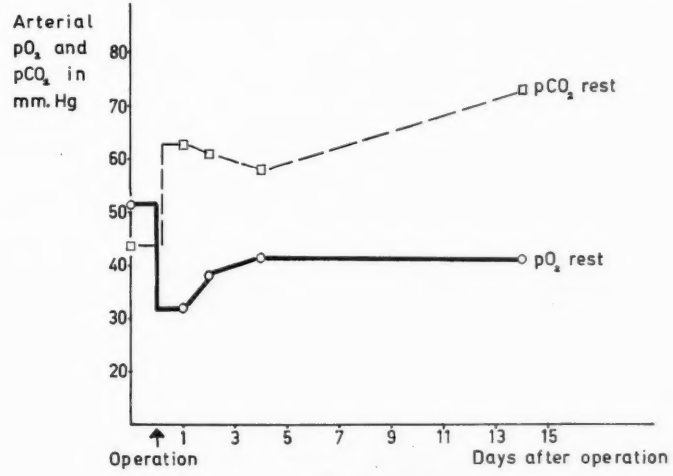


Fig. 3 (Case 5).

Case 5.—A 37-year-old man had a left tuberculous empyema with a bronchopleural fistula. Maximum breathing capacity was 22 L. per minute. The pressure in the pulmonary artery was high. A pronounced oxygenating insufficiency was noted before operation. A left

extrapleural pneumonectomy was performed. After the operation the patient was markedly anoxic with carbon dioxide retention (Fig. 3). He survived this operation and one and one-half months later he had a left thoracoplasty with removal of the first to eighth ribs. The patient was still a respiratory cripple following the operation as he was before, but he now had negligible amounts of sputum compared to the amounts before surgery. This patient had the poorest pulmonary function of any who lived following the operation. One year later this man could walk slowly. His maximum breathing capacity was 18 L. per minute. The resting arterial pO_2 was 59.3 mm. Hg and pCO_2 was 58.1 mm. Hg. Sputum and gastric washings were negative on guinea-pig test.

Conclusion.—After pneumonectomies a considerable decrease in the arterial oxygen tension is observed. The anoxia has been found to last longer than earlier described. Even in good-risk patients the preoperative arterial oxygen tension was first reached after one week, whether the pneumonectomy was done under a thoracoplasty (Fig. 1) or done without thoracoplasty (Fig. 2). In cases of pneumonectomy, impaired ventilation due to pain seems to be the main cause of the low arterial oxygen tension. The phrenic nerve is never divided after a pneumonectomy, since the contralateral diaphragm is thought to function more efficiently if the diaphragm on the operated side is not paralyzed.

II. Segmental Resection Without Thoracoplasty.—

Only one case was investigated in which the resection of the apical segment of the right lower lobe was performed without a thoracoplasty.

CASE 6.—The patient was a 36-year-old man. He had a pneumothorax on the left side. The following values were found:

	Before Operation		12 Days After Operation	
	At rest	Hyp. vent.	At rest	Hyp. vent.
pO_2	90.0	83.5	83.7	82.5
pCO_2	38.9	39.1	42.9	43.1

III. Resections of Lobes and Segments With Extrapleural Plombage.—

In two patients with resection of a lower lobe, an apical extrapleural plastic plomb was inserted to prevent overdistention and to improve the ventilation by bringing the upper lobe down to the diaphragm. These are described in Cases 7 and 8.

CASE 7.—The following values were found:

	Before Operation		2 Weeks After Operation	
	At rest	Hyp. vent.	At rest	Hyp. vent.
pO_2	77.5	99.6	77.0	75.1
pCO_2	45.4	32.8	45.8	35.5

CASE 8.—The following values were found:

	Before Operation		2 Weeks After Operation	
	At rest	Hyp. vent.	At rest	Hyp. vent.
pO_2	73.7	80.4	80.8	89.0
pCO_2	32.0	35.1	41.6	36.1

CASE 9.—A 23-year-old woman underwent a right upper lobectomy combined with extrapleural plombage. The values are shown in Fig. 4. Six months later the plombage had to be removed due to infection which caused a bronchial fistula.

Conclusion.—In only Case 8 could a good hyperventilation test be performed two weeks after the operation. The extrapleural plastic plombage in combination with lobectomy will prevent paradoxical movement of the chest wall. Still a considerable impairment of the arterial oxygen tension was found during the first few days (Case 9, Fig. 4) due mainly to pain and atelectasis.

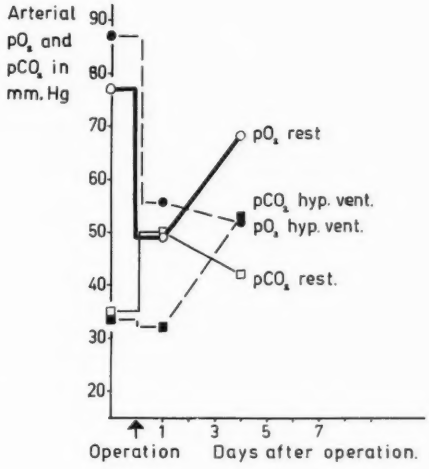


Fig. 4 (Case 9).

IV. Resections of Lobes and Segments Under Old Thoracoplasties.—In this group data were collected on sixteen patients. Only two of these patients could perform a voluntary hyperventilation with an increase of the arterial oxygen tension of more than 5 mm. Hg 9 and 12 days, respectively, after the operations. The preoperative arterial oxygen tension was reached between one and two weeks in nine of sixteen patients. In four cases the arterial pO_2 was still between 2 and 5 mm. Hg lower than before operation. In three cases a lower value of more than 5 mm. Hg was encountered during this period than before operation. In one patient the arterial oxygen tension was only 54.2 mm. Hg on the ninth day after operation as compared to 81.8 mm. Hg before operation. This patient had developed a bronchial fistula.

Five cases with different degrees of impaired pulmonary function are described.

Case 10.—In a 38-year-old man, under a four-rib thoracoplasty, a resection of the right upper lobe plus the apical segment of the lower lobe, plus the fifth to the seventh ribs was performed. Maximum breathing capacity was 90 L. per minute. The tensions are shown in Fig. 5. A pronounced decrease of arterial oxygen tension was found for only a few days, and by the fifth day the preoperative value was already reached.

Case 11.—A 45-year-old man who had had a seven-rib, right-sided thoracoplasty and a left three-rib thoracoplasty now underwent a resection of the posterior segment of the left upper lobe and a wedge of the apex of the lower lobe plus the fourth and fifth ribs.

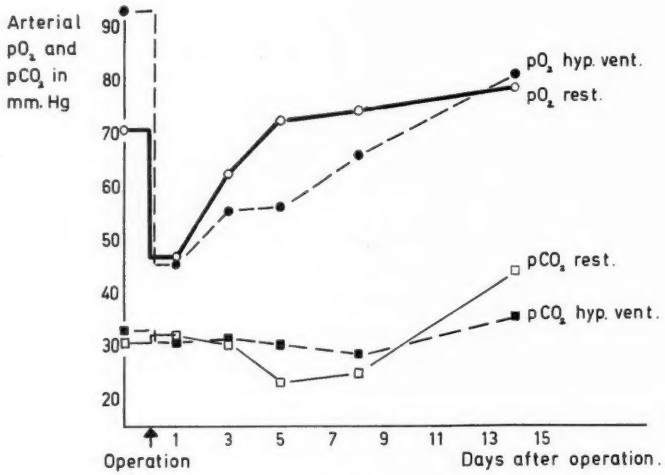


Fig. 5 (Case 10).

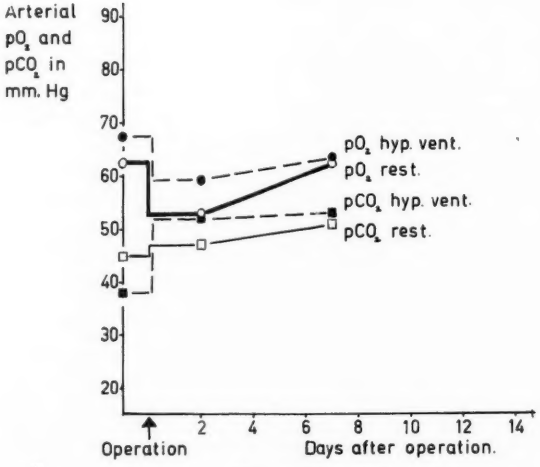


Fig. 6 (Case 11).

Maximum breathing capacity was 49 L. per minute. After one week the preoperative arterial oxygen tension was reached (Fig. 6).

CASE 12.—A 30-year-old woman had a right five-rib thoracoplasty and left extrapleural plombage. Resection of the apicoposterior and anterior segment of the left upper lobe under the plombage was performed. After two weeks the preoperative arterial oxygen tension was first reached (Fig. 7). Three months later a resection of the right upper lobe and lateral segment of the middle lobe was performed without postoperative complications.

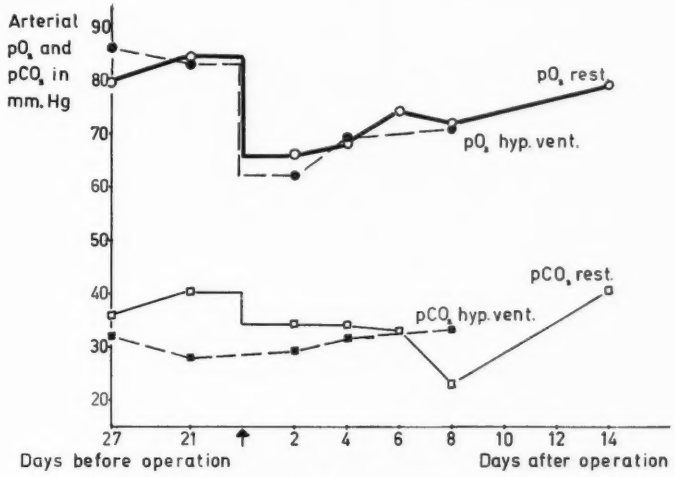


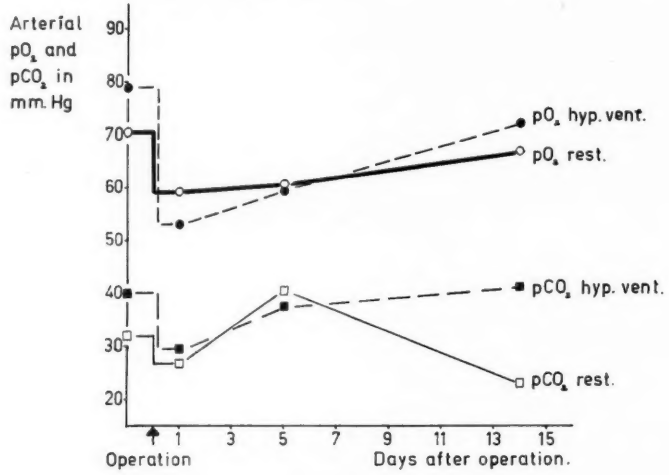
Fig. 7 (Case 12).

CASE 13.—A 27-year-old woman had a left upper lobectomy with decortication of the lower lobe performed under a five-rib thoracoplasty. One and one-half years later the right upper lobe, a wedge from the apical segment lower lobe, and the fourth and fifth ribs were resected under a three-rib thoracoplasty. At the time of the last operation (right upper lobectomy) the maximum breathing capacity was 55 L. per minute. The arterial oxygen tension had not reached the preoperative value fourteen days after the operation (Fig. 8, *A*). The x-ray picture of this patient one month and twenty-two days after operation is shown in Fig. 8, *B*. One year later she was well and sputum negative.

CASE 14.—A 34-year-old man with a left six-rib thoracoplasty had a resection of the right upper lobe plus a wedge resection of the apical segment of the lower lobe resection of the fourth to sixth ribs performed under a 3-rib thoracoplasty. Maximum breathing capacity was 44 L. per minute. Three days before surgery there was a short period of pulmonary edema with a simultaneous drop in arterial oxygen tension (Fig. 9). The day after this episode, when the patient felt better, the resting arterial oxygen tension had reached its original value again. However, seventeen days after the operation the original value had not been reached (Fig. 9).

Conclusion.—After resection under a thoracoplasty in a patient with good pulmonary function (Case 10, Fig. 5) the arterial oxygen tension reached the preoperative value on the fifth day after surgery. In some cases with advanced bilateral disease the arterial oxygen tension had not yet reached the preoperative value after fourteen postoperative days (Case 13, Fig. 8, and Case 14, Fig. 9).

V. Resections of Lobes and Segments With Simultaneous Thoracoplasty.— Six patients were included in this group. There were four cases of unilateral disease, in three cases only one segment was removed in contrast to Group IV. In spite of the good pulmonary function in Group V, the preoperative



A.

B.

Fig. 8 (Case 13).

arterial oxygen tension was reached in only one patient in one to two weeks postoperatively. The simultaneous thoracoplasty with paradoxical movement of the chest wall, as well as the increased pain causing impaired ventilation can explain the prolonged postoperative lowering of the arterial oxygen tension. In

two cases an increase of more than 5 mm. Hg in arterial oxygen tension was encountered during hyperventilation after ten and nineteen postoperative days, respectively.

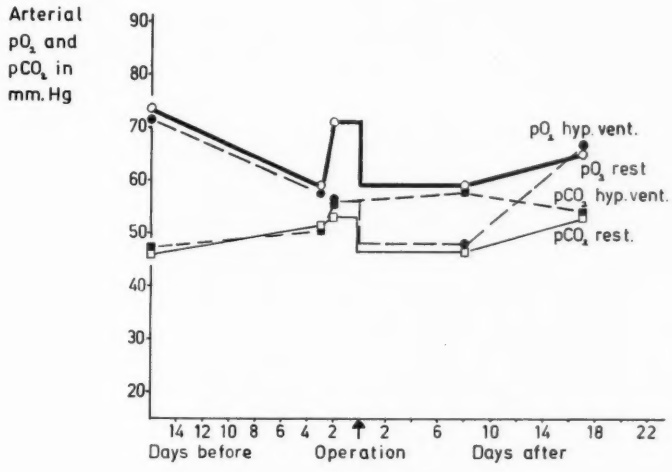


Fig. 9 (Case 14).

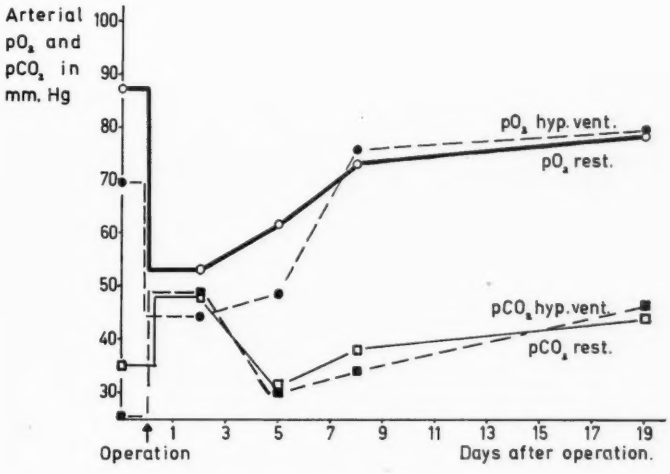


Fig. 10 (Case 15).

CASE 15.—A 27-year-old man had a resection of the apical segment of the right upper lobe plus decortication and removal of the posterior ends of ribs two to five. The preoperative arterial oxygen tension value was not reached after nineteen days (Fig. 10).

CASE 16.—A 64-year-old man had a resection of the apical segment of the right upper lobe and of the posterior ends of ribs two to six. He had normal preoperative values but a pronounced decrease of the arterial oxygen tension after the operation (Fig. 11). A normal response to hyperventilation was found.

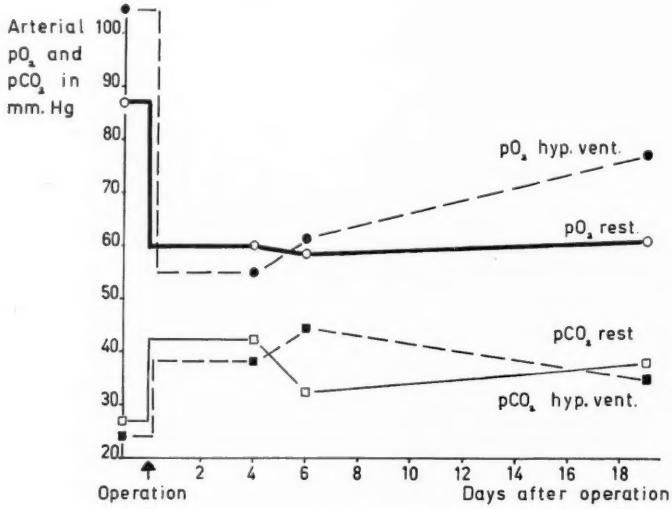


Fig. 11 (Case 16).

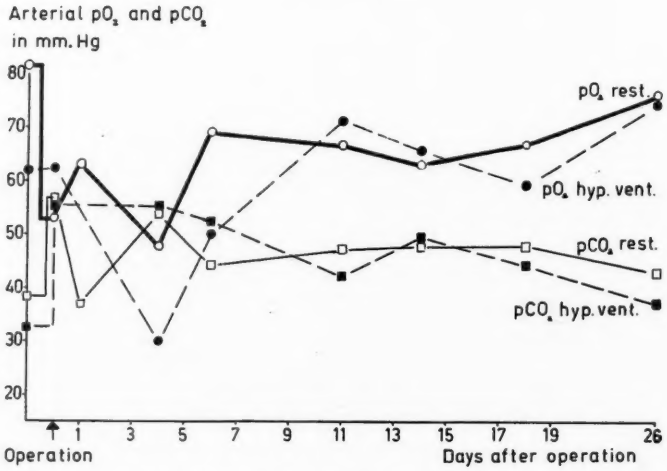


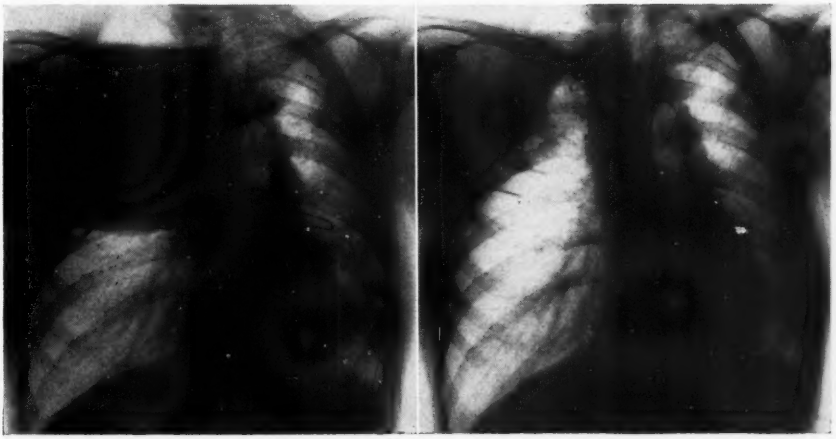
Fig. 12 (Case 17).—A 25-year-old man had a right 5-rib thoracoplasty with an apicolysis performed.

Conclusion.—It has been observed that if a thoracoplasty is performed at the same time as a resection a prolonged decrease in the arterial oxygen tension is encountered.

VI. Thoracoplasty.—

CASE 17.—A 25-year-old man had had bilateral pneumothorax. A five-rib thoracoplasty with apicolysis on the right side was performed. Maximum breathing capacity was 116 L. per minute. Bronchospirometry showed 70 per cent oxygen uptake on the right side. The arterial oxygen tension did approach the preoperative value after twenty-six days (Fig. 12). Fig. 13, *A* shows an x-ray view of the patient before operation. Fig. 13, *B* is an x-ray view eleven days after operation showing a hematoma in the apicolysis space with a compression atelectasis. Twenty-three days after the operation the hematoma had been absorbed and the lung areated (Fig. 13, *C*).

A.



B. *C.*

Fig. 13 (Case 17).—*A*, X-ray view of the chest before operation. *B*, Roentgenogram of the chest eleven days after operation. Hematoma in the apicolysis space. *C*, Roentgenogram of the chest twenty-three days after operation; the hematoma has been absorbed.

Conclusion.—In this case of a five-rib thoracoplasty with apicolysis, a considerable decrease in the arterial oxygen tension was found during the postoperative period. Only after twenty-six days did the arterial oxygen tension begin to approach the preoperative level. Pain, paradoxical movements of the chest wall, and compression atelectasis, all contributed to the decrease in arterial oxygen tension. After about one month when the perfusion of nonventilated areas had diminished and ventilation had improved the normal values were first approached.

DISCUSSION

After all thoracic operations a decrease in the arterial oxygen tension has been found. The measurement of arterial carbon dioxide tension during the postoperative period has shown significant changes only in those patients with advanced disease who had preoperative retention of carbon dioxide. In this group it may be of value to detect the increased carbon dioxide tension early when a tracheotomy may be performed. This will diminish the dead space by approximately 100 ml., thereby improving the alveolar ventilation. In three cases of ventilatory insufficiency, arterial carbon dioxide tensions of 95.6, 117, and 124.5 mm. Hg, respectively, were reached. In one of these patients the pCO_2 dropped 35 mm. Hg after the tracheotomy, but all 3 patients died. The samples taken during voluntary hyperventilation have merely shown that it usually takes at least two weeks after the operation before the patients can increase the arterial oxygen tension during hyperventilation.

A decreased arterial oxygen tension the first few days following surgery has been found in pneumonectomies as well as in resections of lobes and segments with and without thoracoplasties. This immediate decrease in arterial oxygen tension is due to impaired ventilation. Pain, paradoxical movements of the chest wall, and atelectasis are all factors causing an impaired ventilation. In cases of pneumonectomy usually no atelectasis is present. In cases with simultaneous thoracoplasty, however, atelectasis, and paradoxical movements of the chest wall may be significant enough to cause a prolonged decrease in the arterial oxygen tension. From a functional point of view we therefore follow the policy of performing a first stage thoracoplasty with removal of the third to fifth upper ribs with apicolysis to the aortic arch or hemiazygos vein, respectively, in cases where lung tissue corresponding to a lobe or more is going to be resected. Then the resection is performed after two to three months.

There is, however, in many cases a need to diminish the thorax at the time of resection. After resection of a lower lobe we have done an extra pleural plastic plombage over the apex to dislocate the upper lobe down to the diaphragm in order to obtain the best possible ventilation. In most cases where the thoracic cavity is diminished I (Björk) now use a thoracoplasty with resection of a small segment of the posterior ends of ribs two to five, two to six, or two to seven according to the amount of lung tissue resected. Small drill holes are made in the posterior ends of these ribs. Then drill holes are made as far back as possible in the uppermost rib which is not resected.

Heavy stainless steel sutures through the holes will fix the posterior ends of the divided ribs which are bent in their cartilages to the uppermost intact rib and make a stable roof. The first rib is either left in place or divided in front and at the back and sutured to the fifth transverse process. An apicolysis is performed. In this way we hope to prevent the paradoxical movement of the chest wall. Furthermore with this method a thoracoplasty of a desired size can be obtained and will remain fixed. The earlier tailoring thoracoplasty was usually found to be much smaller than expected when re-examined one year after the operation.

SUMMARY

In thirty-two patients with different degrees of pulmonary reserve, pre- and postoperative studies of the arterial oxygen and carbon dioxide tension have been made.

1. After pneumonectomy a decreased arterial oxygen tension was found for one week whether the resection was done under an old thoracoplasty (no paradoxical mediastinal movements) or not.

2. In patients where a resection of lobes and segments under a thoracoplasty was performed the preoperative arterial oxygen tension was reached within two weeks in nine of sixteen cases.

3. In a patient with a thoracoplasty and apicolysis a prolonged period of decreased arterial oxygen tension was found. The paradoxical movements of the chest wall in combination with some atelectasis are thought to be the main source of this finding.

4. If a resection of lobes and segments was performed at the same operation as a thoracoplasty, a prolonged impairment of the arterial oxygen tension was found. To exclude the paradoxical movement of the chest wall a thoracoplasty is now performed in which the ribs are fixed to the uppermost intact rib by sutures through drill holes.

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CARBON DIOXIDE IN ANESTHETIC ATMOSPHERES AS MEASURED BY THE LISTON-BECKER (INFRARED ABSORPTION) GAS ANALYZER

Preliminary Report

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As "OPEN" thoracic procedures become more and more common, the attention of anesthesiologists, thoracic surgeons, and research workers has been focused on the problem of adequate ventilation of the lungs of patients during the administration of anesthetic agents. Investigation of the problem has shown that some anesthetic techniques, most anesthetic agents,[1-3] and some positions of the patient[4] necessary for the surgeon to accomplish his task interfere with adequate ventilation. This causes hypoxia and/or hypercarbia with consequent upset of the acid-base balance of the body, which is reflected in the blood pH and pCO_2.

The alert anesthesiologist has many signs upon which he depends to guide him in preventing serious damage to his patient due to hypoxia. However, it has been shown by Comroe and Botelho[5] that groups of competent observers have been unable to recognize hypoxia in an anesthetized patient until the blood oxygen saturation drops to *70 per cent or below*.

The patient who has excessive accumulation of carbon dioxide in the body during the administration of an anesthetic does not have a sign as obvious as cyanosis to warn us. Blood pressure elevation, variations in the pulse, changes in the respiration, and, of course, muscle twitching occur relatively late. Recent studies made using the mass spectrometer by Buckley and by Miller, with their respective co-workers[6, 7] have shown that with common anesthetic techniques, allowing the patient to breathe spontaneously and unassisted, carbon dioxide is retained in the patient more than the anesthesiologist realizes, or would allow, if he were able to judge by clinical signs. Admittedly, the mass spectrometer is bulky, highly technical to operate, and far beyond the cost most hospitals or anesthesiologists could afford.[7]

Recently, Bardeen[8] has been working with a small, inexpensive carbon dioxide analyzer for testing anesthetic mixtures. This analyzer requires a large sampling volume (500 to 700 ml.) which limits its continuous use, especially in closed anesthesia systems.

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"Open" thoracic procedures constitute 95 per cent of the major operations in this hospital. In an effort to determine the best means of administering anesthetics for this type of operative procedure, it became apparent that some practical means of determining the variations in the patient's acid-base balance during the course of the operation was desirable. For this purpose we have been using the Liston-Becker Gas Analyzer (infrared absorption) to measure the carbon dioxide in the respired atmosphere of the patients. Efforts were made to determine the best method of sampling in order to reflect accurately the changes in acid-base balance in the patients' blood as they may occur.

METHOD

This paper deals with the first forty cases and some of the difficulties encountered. The patients were all given a dose of 0.3 mg. of scopolamine one hour before induction of anesthesia. Anesthesia was begun with 300 to 500 mg. of Pentothal Sodium and 18 to 24 mg. of tubocurarine. After a short period of breathing (or inflating the lungs) with 100 per cent oxygen, a cuffed endotracheal tube or Carlen double lumen catheter was put in place. Maintenance of anesthesia was with a flow of 3.5 L. per minute of nitrous oxide and 1.5 L. per minute of oxygen with a to-and-fro canister in place. Assisted respiration was used as soon as the patient was turned to the lateral position for the surgical procedure. Controlled and/or artificial respiration were used while the pleura was open, and further barbiturate or relaxant drug was used as needed.

The Liston-Becker Gas Analyzer (infrared absorption) (Fig. 1) consists of two parts: the "pick-up" and the control units. The "pick-up" unit is sealed and may be "pressured" up to five pounds air pressure to make it safe for use with explosive anesthetic mixtures. This unit must be located close to the patient's head. A bracket may be made to fit any ordinary operating table (Fig. 1). The "pick-up" unit has interchangeable sampling cells. One measures the carbon dioxide as all of the anesthetic mixture passes through the cell, either as it enters or leaves the face mask or endotracheal tube. For our studies we have been using another type of cell, the "nasal catheter cell." A small, continuous sample is drawn from the anesthetic mixture by means of slight suction, sufficient to obtain the maximum deflection of the recording unit. The sample is obtained through a small catheter (Becton-Dickinson No. 442T polyethylene tubing or a No. 5F ureteral catheter). The tip of this catheter can be placed in any part of the anesthetic atmosphere from the bronchi to the breathing bag. This method of sampling the anesthetic mixture provides more latitude in the different areas which may be checked, and it is more adaptable to different anesthetic techniques.

The control unit is connected to the "pick-up" unit by a cable, and may be placed at a safe distance, outside the operating room if necessary, when explosive mixtures are used. The carbon dioxide in the sample causes a deflection of a milliampere meter hand, which can be read directly or permanently recorded by means of an Esterline-Angus recorder or any of the various

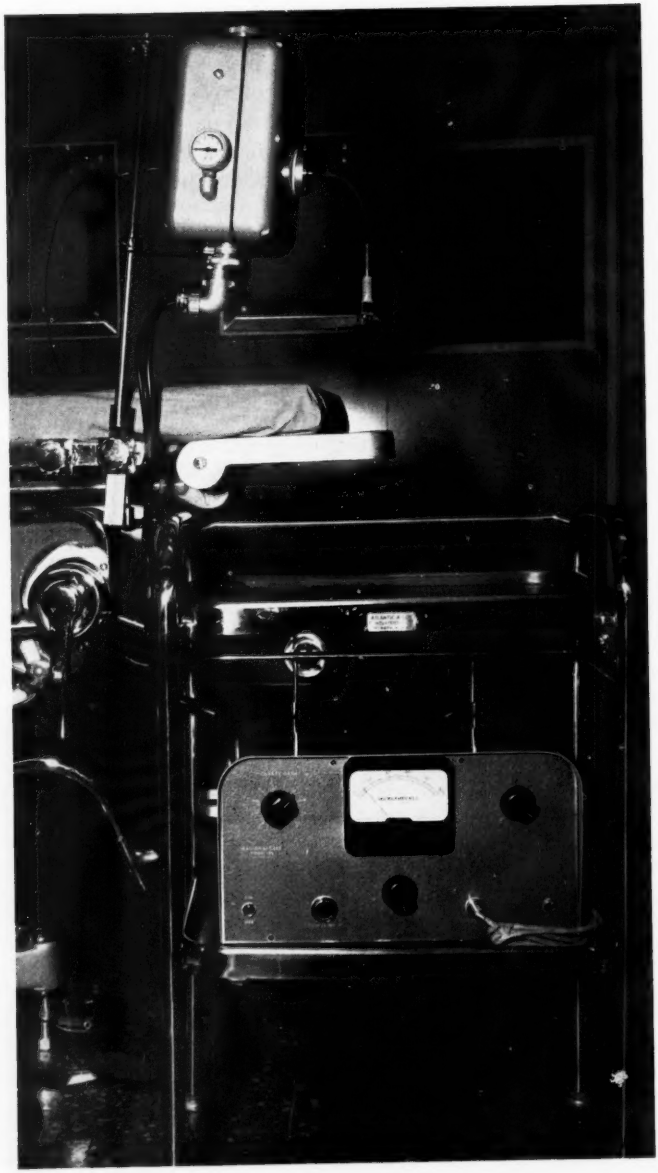

Fig. 1.

direct writing electrocardiographs (such as those of the Sanborn variety). There is a "lag" of 0.1 second in the recording system. Water *vapor* and anesthetic gases have no recordable effect on the readings.[9]

It is necessary to turn the instrument on at least one hour before use, and it is more accurate and requires less frequent calibration if allowed to warm up overnight before using. The range of the catheter cell is 0 to 15 per cent carbon dioxide. The instrument is calibrated by the use of three known mixtures of carbon dioxide. We have set air as the zero concentration of carbon dioxide and have three tanks of carbon-dioxide-oxygen (approximately 2 per cent, 5 per cent, and 10 per cent) for calibration. The mixtures were previously analyzed by the Scholander method.[10]

It is necessary to recheck the calibration with two of the known mixtures at least every hour. If droplets of moisture, sputum, or other materials are sucked into the sampling cell it is necessary to clean it by drawing ether and/or carbon tetrachloride through it and permitting it to dry thoroughly.

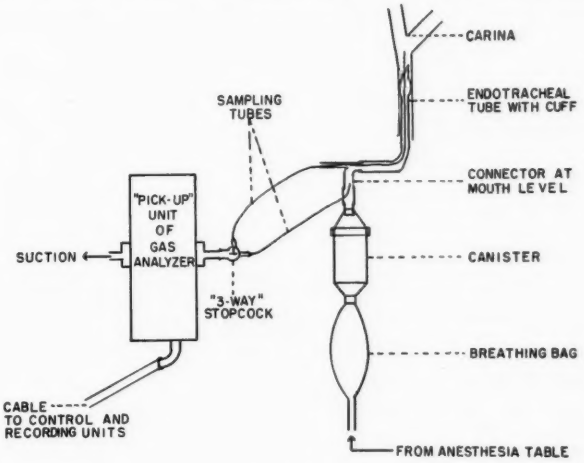

Fig. 2.

By means of a three-way stopcock (Fig. 2) and two sampling catheters, tidal samples could be drawn from two areas for comparison, within the space of one or two respirations.

The blood samples were drawn from the pulmonary vein or occasionally, when the thorax was not open, or the surgeon too busy, from a peripheral artery. The whole blood carbon dioxide content was determined by the Van Slyke manometric method. The whole blood pH was determined by the glass-electrode pH meter, kept at a constant temperature of 37° C. Some of the gas samples were drawn through the infrared sampling cell into gas-sampling tubes, and these samples were analyzed by the Scholander method to check the accuracy of the instrument. These analyses correlated with the readings on the gas analyzer within 0.06 per cent.

"Alveolar" gas samples were taken by clamping the rubber tubing at the junction of the endotracheal tube connector and the to-and-fro canister, thus diminishing mixing caused by the tidal flow of gas, and drawing the sample from the endotracheal tube, bronchi, and the more peripheral areas of the lung.

This "alveolar" reading is much more stable and accurate (as checked by blood analyses) (Table I) when all breathing effort can be temporarily stopped, as during controlled or artificial respiration.

RESULTS

The "alveolar" samples averaged about 33.9 per cent higher in carbon dioxide content than the tidal samples taken from the same area, and most of the time approximated more closely the variations in the pH and pCO_2 of whole blood.

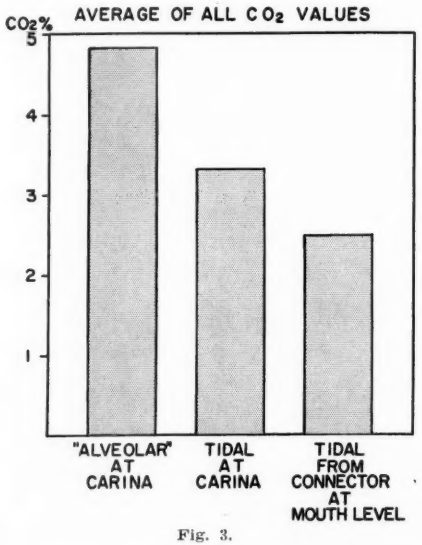

Fig. 3.

Tidal gas samples drawn from the endotracheal tube connector, as it projects from the mouth, average about 24.8 per cent less carbon dioxide than tidal samples drawn from the carinal area (Fig. 3).

DISCUSSION

Forty patients have been studied in this manner and it has been found that the tidal samples drawn from the region of the carina of the trachea are consistently higher (Table I), and at the height of the curve probably more nearly approach true alveolar air. Our "alveolar" samples more nearly reflect the actual concentration of carbon dioxide of whole blood than do samples drawn from "higher" parts of the pulmonary-anesthesia system. (Table II.)

It must be pointed out that our "alveolar" samples are "spot" samples. Fowler and Comroe[11] state that "a single 'spot' sample of expired gas is not necessarily an average or representative specimen of lung gas." Some of our "alveolar" samples showed a carbon dioxide content in excess of that of the

TABLE I

CASE NO.	AV. OF TIDAL SAMPLES CO_2% TAKEN AT MOUTH LEVEL	AV. OF TIDAL SAMPLES CO_2% TAKEN AT CARINAL LEVEL	AV. OF "ALVEOLAR" SAMPLES, CO_2% TAKEN AT CARINAL LEVEL
1	3.50	4.32	5.73
2	1.30	1.87	3.46
3	3.9	4.97	8.60
4	2.68	2.94	4.66
5	4.30	4.46	6.46
6	2.22	2.38	4.01
7	1.89	3.07	4.40
8	1.10	1.73	3.49
9	2.03	2.90	4.17
10	—	4.90	6.08
11	1.49	2.10	3.95
12	—	2.97	4.70
13	—	2.42	3.06
14	1.70	1.94	4.24
15	1.42	3.14	4.70
16	—	5.46	7.35
17	—	2.97	4.50
18	—	2.42	3.10
19	—	5.90	7.60
20	—	3.27	5.06
21	—	3.20	3.58
22	—	2.15	3.77
23	—	2.82	4.74
24	—	4.75	6.10
25	—	1.25	3.90
26	—	1.82	3.56
27	—	2.70	3.95
28	—	2.15	4.30
29	—	2.75	3.90
30	1.65	2.70	2.70
31	1.90	—	3.00
32	1.65	—	2.95
33	1.70	2.95	3.20
34	0.95	2.15	3.00
35	2.15	4.95	7.00
36	4.96	5.55	7.50
37	2.70	3.70	4.50
38	3.95	4.50	5.35
39	4.85	5.50	7.40
40	3.25	4.50	6.50

carbon dioxide of arterial blood drawn from the pulmonary vein simultaneously with the "alveolar" sample. This would indicate that this type of sample is drawing gas from areas of lung that have recently been inadequately ventilated or to which the circulation has been impaired for a time. This could easily be due to mechanical factors, such as retraction by the surgeon, or compression of the lung by packs, instruments, or hands.

The readings of the carbon dioxide analyzer are very sensitive to mixing of the gas in the sampling area caused by inequalities in the tidal flow. Overdistention of the anesthetic breathing bag, causing continuous positive pres-

sure (even of a very mild degree, 1 to 3 cm. water pressure), causes lowering of the carbon dioxide reading. This indicates inadequate ventilation under these conditions. Wilhelm, Orth, and Waters[12] found that "a half filled bag, simply hanging down from an endotracheal tube connection will create a condition of constant positive pressure in the system. Further pressure is created if a hand 'rests' on the bag, or the bag is fully distended." These findings emphasize the fact that the anesthesiologist must release all pressure from the breathing bag between inflations of the lung, not only to allow diastolic filling

TABLE II

CASE NO.	BLOOD SAMPLE NO.	TIDAL SAMPLE AT MOUTH LEVEL CO_2 IN MM. Hg TENS	TIDAL SAMPLE AT CARINA CO_2 IN MM. Hg TENS	"ALVEOLAR" SAMPLE AT CARINA CO_2 IN MM. Hg TENS	CO_2 IN VOL. % OF WHOLE BLOOD	pH WHOLE BLOOD
14	1.	8.88	18.87	29.60	48.19	7.53
	2.	11.84	12.58	22.94	45.37	7.62
	3.	8.88	14.06	25.90	44.56	7.53
	4.	16.65	24.42	37.00	49.78	7.47
	5.	14.06	21.22	34.06	46.75	7.40
15	1.	—	38.48	42.92	54.30	7.35
	2.	—	19.98	28.49	48.30	7.40
	3.	10.36	10.36	23.31	43.90	7.57
	4.	—	—	41.00	48.60	7.26
	5.	—	—	34.69	45.00	7.57
24	1.	—	—	60.40	48.31	7.38
	2.	—	—	33.30	47.75	7.36
	3.	—	—	37.00	45.43	7.43
	4.	—	—	51.80	47.22	7.41
	5.	—	34.78	42.92	49.00	7.52
26	1.	—	14.06	31.82	56.54	7.26
	2.	—	8.88	26.64	49.81	7.36
	3.	—	11.84	23.68	44.00	7.40
	4.	—	16.06	26.64	48.64	7.32
	5.	—	15.42	26.64	49.77	7.38
	6.	—	14.06	22.06	45.54	7.40

of the heart, as pointed out by Best and Taylor,[13] but to allow free outflow of alveolar gases. Sudden compression of the lung during retraction by the surgeon or his assistants causes a high "peak" reading, momentarily, followed by a lower series of "peaks" if the retraction is maintained and other areas of the lung are not sufficiently ventilated to compensate for it. The tidal readings taken during the periods of inadequate ventilation may show a "low level" of carbon dioxide, while an "alveolar" reading taken at this time, especially after a more vigorous inflation, will indicate that carbon dioxide is "accumulating" in the blood and body. This "alveolar" reading cannot be taken as a true average due to the conditions pointed out above.

This instrument has been valuable to us in following the changes in the carbon dioxide content of the anesthetic atmosphere during periods of assisted, artificial, and controlled respiration while administering anesthetics for "open" thoracic procedures. Once the instrument is set up and calibrated, it is relatively simple to place the sampling catheters and a continuous record can be taken. It is necessary to be vigilant in not allowing secretions or water *droplets* to be sucked into the sampling cell. This fact has probably been in-

strumental in causing the anesthesiologist to perform more frequent and effective tracheobronchial aspirations. It has been noted that in this series of patients, postoperatively there has been less than the usual amount of secretions and consequently less atelectasis.

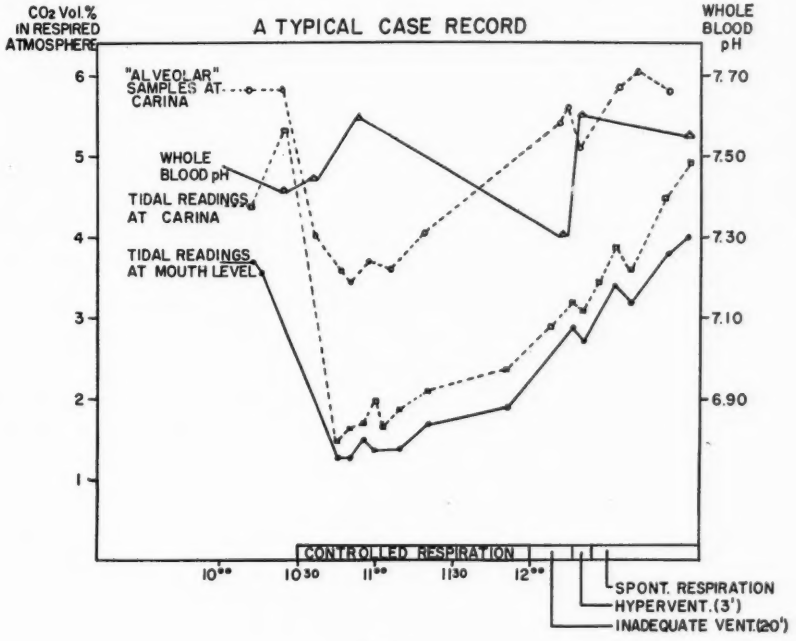

Fig. 4.

The curves, if the carbon dioxide values and the blood pH values are inscribed graphically, follow a pattern as shown in Fig. 4. It will be noted that tidal values of carbon dioxide are sufficient to follow the *trend* of the whole blood pH and are preferred for routine use giving a good indication of changes in acid-base balance. The tidal readings at the mouth level are much easier to control, due to less accumulation of secretions in this area. However, variations in mixing due to a distended breathing bag, or to inadequate relaxation of the anesthesiologist's hand on the bag may cause errors in samples from this area.

SUMMARY

1. A method of sampling anesthetic atmospheres and recording the variations in carbon dioxide content has been described.

2. Forty cases have been studied to determine the variations in the carbon dioxide content when samples are drawn from different areas in the "pulmonary-anesthesia system."

3. Some causes of error are noted.

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THYMIC CYSTS

A Review of the Literature and Report of Two Cases

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INTRODUCTION

ALTHOUGH both the neck and the mediastinum are fairly common sites for cystic lesions of various types, cysts arising in the thymus gland are encountered most infrequently. Virtually all cysts of thymic origin reported in the early literature were found in syphilitic infants and were identified exclusively at necropsy. Thymic cysts due to syphilis have rarely been described during the present century. However, within the last decade a different variety of thymic cyst has been observed, and lesions of this type have been resected from both the neck and the mediastinum. Excision of tumors of the thymus gland has become commonplace in recent years and large series of cases treated in this manner have been reported by Seybold and his associates,[31] Blalock,[2] and others,[18, 23, 24] yet only a few nonsyphilitic thymic cysts have been recorded in the literature.

Blades[1] and Laipply,[21] in extensive reports of mediastinal tumors and cysts, do not mention this lesion. The rarity of cysts of the thymus is attested by the fact that the literature contains reports of only eight which have been resected (Table I). Of these, four were removed from the neck, two from the mediastinum, and two by a combined cervical and mediastinal approach. None of these eight patients was syphilitic. The purpose of this report is to review the literature concerning thymic cysts and to record two such lesions which were excised.

HISTORICAL REVIEW

Lieutaud[22] described suppurative changes in the thymus, in 1832, and he noted other changes due to syphilis during post-portem examination of the two young male adults in whom the lesions were found. However, he related the alterations in the thymus to the caseous pulmonary tuberculosis present in each patient. Dubois,[7] in 1850, described cystic alteration of the thymus in three newborn infants, who died of congenital syphilis. He believed these cysts to be due to syhilitic suppuration. He stated that the changes were pathognomonic of syphilis and suggested that in Lieutaud's cases the alterations were due to syphilis rather than tuberculosis. Many authors[4, 8, 9, 12, 16, 19, 20, 26, 28, 29, 30, 32, 36, 37, 39] subsequently described cystic changes in the thymus. Most of these early reports appeared in the European literature and all the studies were made from

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TABLE I. BENIGN NONSYPHILITIC THYMIC CYSTS

AUTHOR	DATE	AGE	SEX	LOCATION	SIZE	WEIGHT	DIAGNOSIS	LINING	CONTENTS
Soupalt[34]	1897	18 yr.	F	Mediastinal	6 x 13 cm.	69 Gm.	Necropsy	Tall cylindric cells, lining often irregular	Mucoid material, desquamated cells
Westenryk[39]	1900	51 yr.	F	Mediastinal	"Walnut size"		Necropsy	Ciliated columnar epithelium	Multilocular, milky fluid
Polloson and Piery[28]	1901	1½ yr.	M	Cervical	"Fist size"		Excision (partial) and necropsy	Flat pavement cells	Multilocular with brown fluid
Pezeoller[27]	1929	5 yr.	F	Cervical and mediastinal	1 cm.		Excision (partial)	Cuboidal and cylindric cells	Serous material of brownish color containing granular and cellular debris
Fridjohn[11]	1934	12 hr.	F		"As large as a walnut"		Necropsy	No microscopic examination	Thick glairy fluid, unilocular
Speer[35]	1938	25 yr.	M	Mediastinal	7 x 5 x 1.3 cm.	4-6 Gm.	Necropsy	Simple and pseudostratified columnar epithelium, some ciliated; some areas flattened nonciliated cells	Granular and vacuolated material, multilocular
Kopac[19]	1939	36 yr.	F	Mediastinal	"Like bunch of grapes"	60 Gm.	Necropsy	Flat epithelium-like cells; cubic and cylindric forms seen	Clear red liquid, multilocular

*Case reported in article.

Hyde, Sellers, and Owen[17]	1939	5 yr.	M	Cervical	7 x 5.5 x 3.2 cm.		Excision	Pavement cells	Thick yellowish-brown liquid containing cholesterol crystals; multilocular
Bradford Mahon, and Grow[3]	1947	22 yr.	M	Mediastinal	One lobe 14 x 2.5 x 5 cm.; other lobe 12 x 5 x 7 cm.	74 Gm.	Excision	Appeared to be persistence of epithelial ramifying strands of fetal thymus; cystic distention occurred in these	Multilocular
Smart[33]	1947	53 yr.	F	Cervical and mediastinal	8 x 5 x 5 cm.		Excision	Flat nonciliated cells	Straw-colored fluid, unilocular
Crellin Pugh, and Janton[5]	1950	40 yr.	F	Mediastinal	11 cm.		Excision	Thick layer of fibrous tissue containing cholesterol crystals; no epithelial lining	Thin sanguineous fluid and brick-red amorphous material, unilocular
Heinz[13]	1950	6 yr.	M	Cervical	?	?	Excision	Squamous epithelium	?
Weller, Pearce, and Rapoport[38]	1950	4 yr.	F	Cervical	4.0 x 7.5 x 4.5 cm.		Excision	Flattened cells and stratified squamous epithelium	Cloudy, pale brown fluid, unilocular
Case 1*	1953	21 yr.	M	Mediastinal	6 x 10 cm.		Excision	Low columnar to transitional-like epithelium	Chocolate-brown thick fluid; multilocular cyst
Case 2*	1953	25 yr.	M	Mediastinal	7 x 3.5 x 2.5 cm.		Excision	Cells which appear to be reticulum cells; areas where cells appear more related to epidermoid cells	Amber-colored watery fluid; multilocular cyst

necropsy material. The reported cysts were universally attributed to syphilis of congenital origin. The gland in these cases was generally multicystic, with the individual cysts varying in size from a few millimeters in diameter to "almost walnut size."

In 1901, Polloson and Piery[28] reported the unsuccessful attempt at removal of a fist-sized mass in the right neck of a 1½-year-old boy. The mass was partially removed by morcellation, but the patient died the day of operation.

Pezcoller[27] reported, in 1929, the partial excision of a combined cervical and mediastinal thymic cyst which presented as a mass in the suprasternal notch of a 5-year-old girl. The base of the cyst passed behind the sternum into the mediastinum. The stalk was amputated and the operation discontinued because of hemorrhage. In 1939, Hyde, Sellers, and Owen[17] reported the first complete excision of cervical thymic cyst. The lesion presented as a soft round mass about the size and shape of an ordinary hen's egg just below the angle of the right jaw anterior to the sternocleidomastoid muscle.

The first reports of a mediastinal thymic cyst to be excised at thoracotomy appeared simultaneously in the American and British literature in October, 1947. On Aug. 23, 1946, Bradford, Mahon, and Grow[3] removed a mediastinal thymic cyst, and stated, "We are presenting a thymic cyst in an adult which completely replaces the gland, is not due to syphilis, and is apparently the first cyst of its type and size to be removed successfully." On Sept. 26, 1946, Smart[33] removed a large anterior mediastinal thymic cyst. The patient sought attention because of "tightness in the neck" of one year's duration. This symptom was aggravated by lying down. When the cyst could not be removed through the thoracic approach, a thyroid-type cervical incision was made to complete its removal. Crellin's[5] patient had an anterior mediastinal cyst which was lower in location than the others which have been reported. It presented as a radiopacity overlying the left ventricular configuration. Brown, opaque, moderately thick odorless fluid (125 c.c.) was recovered by aspiration prior to thoracotomy.

EMBRYOLOGY AND ANATOMY

The thymic primordium is an entodermal outgrowth of the third branchial pouch on each side of the midline; the fourth branchial pouch rarely gives rise to some thymic tissue. The lower ends of the third pharyngeal pouch and the detached cervical sinus become solid epithelial bars which unite during the eighth week, become attached to the pericardium, and gradually sink with the latter into the thorax. The primordium has a narrow cufflike lumen and a thick wall consisting of several layers of cylindrical epithelium. The epithelial bud proliferates irregularly so that the lumen normally disappears and anastomosing strands extend into the mesenchyme. The future lobules arise at the ends of these branches. By the end of the third month differentiation into a cortex and a medulla is apparent.[25]

The thymus is a bilobed gland normally situated in the anterior mediastinum. The two lobes are closely joined by connective tissue but are not fused. Each lobe is divided into a number of lobules of varying size. The gland con-

sists of a cortex and a medulla which are usually sharply differentiated by a zone of connective tissue called the Strandberg layer. The cortical substance consists of densely packed masses of small cells which are histologically identical with small lymphocytes, the so-called thymocytes. Although the origin of the thymocytes has not been fully established, the preponderance of opinion is that they are lymphocytes which have migrated into the thymus. As the medulla is approached, these thymocytes diminish in number and they are very sparse in the medulla proper. The latter consists predominantly of reticular cells similar to those found in the reticulum of the cortex. These reticular cells are believed to be derived from the original epithelium and thus differ from most other reticular cells of the body. The reticular cells are said to give rise to Hassall's corpuscles, although the origin of these bodies has also been ascribed to atrophy of terminal blood vessels. Degeneration and hyalinization are common in Hassall's corpuscles; they sometimes become calcified.

Thymic cysts, which appear to be related to branchial and cervical cysts, are usually derived from a remnant of the third branchial pouch. They may be located in any position along a line extending from the angle of the jaw medially to the midline of the neck, thence descending into the anterior mediastinum to the pericardium as far distant as the diaphragm. These lesions are most frequently detected as an anterior mediastinal mass on chest roentgenograms or as a palpable mass above the suprasternal notch. A cervical mass, especially if in the suprasternal notch, may represent an extension of a mediastinal thymic cyst or it may be a solitary lesion confined to the neck.

ETIOLOGY AND CLASSIFICATION

Dubois[7] considered that the presence of pus in the thymus had resulted from a syphilitic inflammation of that organ. Simmonds[32] found great numbers of spirochetes in these cavities. Chiari[4] felt that these were not abscesses but epithelium-lined cysts filled with small thymus cells. He observed that such cysts may also be found in children without evidence of syphilis. Eberle[8] described cysts filled with pus and believed them to be from rests of the thymic duct lumen arrested during fetal development as a consequence of syphilis. Tuve[37] ascribed their origin to medullary necrosis. Kopac,[19] noting the age of his patient (36 years) and the unilateral alterations, rejected the concept of a developmental arrest and ascribed the etiology in his case as due to medullary necrosis, with reticular cell lining of the necrotic area resulting in the formation of a cyst.

Heinz,[13] in his discussion of lymphoepithelial cysts of the neck, defined a branchial cyst as one lined by epithelium and surrounded by a well-developed lymphoid stroma. He believes that branchial cysts are probably derived from a remnant of the thymic duct and suggests that these cysts be called "lateral lymphoepithelial cysts" in preference to the term "branchial cyst," a term which he considers misleading. Hicks[15] has recently observed that thymus tissue is said to occur occasionally in branchial cysts. However, in 100 branchial cysts reviewed for histologic characteristics in his laboratory, no unequivo-

cal thymus tissue was seen. Heinz reported the case involving a 6-year-old boy with a palpable mass in the posterior triangle of the neck at the angle of the jaw. The contents of the cyst were not stated. Papillary projections within the cyst were covered by a stratified squamous epithelium. There was an abundant lymphoid stroma about this, which contained Hassall's corpuscles. Heinz, however, did not use the term thymic cyst in his description. He is of the opinion that these lateral lymphoepithelial cysts are akin to the solid lymphadenomas of the neck, which occasionally do contain squamous epithelium. Heuer and Andrus[14] recorded an anterior mediastinal cyst which showed a configuration on roentgenogram similar to Case 1 reported here. They described it as a "cyst originating in a branchial rest."

Persistent tubular remnants of the third branchial pouch (thymic duct) are thought to undergo hemorrhage or fluid accumulation causing enlargement of the thymus gland. Previously normal chest roentgenograms in several of these cases attest to this enlargement. The cause of fluid accumulation or hemorrhage has not been established. Trauma seems unlikely and infection is not borne out by the clinical course or by histologic examination of the excised gland.

Crotti[6] described (1) the ciliated cyst, which may be derived from several processes within the gland, and (2) the congenital syphilitic cyst. Elsewhere in his text he mentioned "hemorrhagic cysts" due to rupture of terminal blood vessels into a preformed epithelium-lined cavity, a remnant of the thymic duct, and almost always found in congenital syphilis.

Ewing[10] states that these cysts may be derived from (1) the epithelial canals of the embryonal thymus which persist and form one or several small or large cysts, and especially frequent in syphilitic patients, (2) dermoid cysts, (3) invasion and distention of Hassall's corpuscles, and (4) cystic lymphangioma.

Speer[35] observed that thymic cysts may be of five types, developing (1) from embryonal remnants of the thymopharyngeal ducts, the branchial clefts, or the thymic tubules, (2) from sequestration products in pathologic involutions of the gland, (3) from degenerating Hassall's corpuscles, (4) from lymph vessels, blood vessels, or connective tissue in various stages of thymic development, hyperplasia, or involution, and (5) from neoplastic processes in the lymphoid cytoreticular or connective tissues.

We believe that thymic cysts may be divided logically into three groups: (1) congenital, (2) inflammatory, and (3) neoplastic. The nonneoplastic, noninflammatory cysts of the thymus reported here are probably congenital in origin, in that a congenital defect may have been present in the form of a patent thymic or thymopharyngeal duct persisting until such time as fluid or hemorrhagic distention occurred. Why this happens is not known. Cysts resulting from infection appear invariably to be due to syphilis. Although rather common in the past, thymic cysts of this etiology are now rarely encountered. Cysts occurring in thymic neoplasms can probably be attributed to degeneration and necrosis of the tumor.

CASE REPORTS

CASE 1.—B. C. F., a 21-year-old white man, was in good health until he developed a left lower lobe pneumonia in January, 1952. Chest roentgenograms revealed a pneumonic infiltration in the left lower lung field and a bilaterally widened superior mediastinal shadow (Fig. 1). He recovered uneventfully from the respiratory infection, but the superior mediastinal shadow persisted. There was no history to suggest either congenital or acquired syphilis.

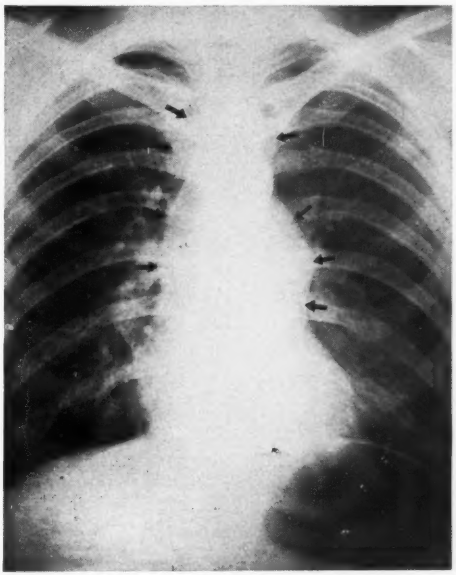


Fig. 1.—(Case 1.) Posteroanterior roentgenogram demonstrating the mediastinal mass.

Physical examination and laboratory studies were normal. The Kahn test was negative. The anterior mediastinal mass, which overlay both hilar regions, extended upward to the level of the sternal notch. The mass could not be clearly delineated in the lateral view. A chest film made in October, 1951, had also shown an abnormal right superior mediastinal shadow. Considerable enlargement of the mass to the left during the intervening three months was seen. Angiocardiographic studies did not demonstrate any opacification of the lesion. Bronchoscopic examination showed the tracheobronchial tree to be normal in configuration, with no evidence of bronchial compression. Bronchograms established the presence of bilateral bronchiectasis with most marked involvement on the left. On March 24, 1952, a left thoracotomy was performed through a posterolateral incision. A free pleural space was encountered. The bronchiectatic left lower lobe was first resected.

Attention was then directed to the mediastinal tumor. It was found to be completely cystic in nature. The presenting surface appeared to be roughly oval in shape and measured about 4 cm. in diameter. The mass presented over the left auricle and the phrenic nerve and its accompanying vessels coursed over the cyst at the junction of its middle and posterior thirds. The phrenic nerve was adherent to the capsule. The cyst overlay the ascending aorta and the aortic arch, extending outward to the left to the point of take-off of the subclavian artery (Fig. 2). The pleura was stripped from the capsule with relative ease. While the pleura was being dissected from the capsule of the mass, the lesion was perforated and some chocolate-colored material extruded. The cyst was found to extend medially, crossing the

ascending aorta and projecting into the right chest. A necklike prolongation extended upward, coursing in a generally anterior direction so that at the sternal notch it lay between the innominate vein and the posterior surface of the sternum. This necklike prolongation ascended into the neck about 2.5 cm. above the superior margin of the manubrium. The cyst was removed in toto through the thoracic approach.

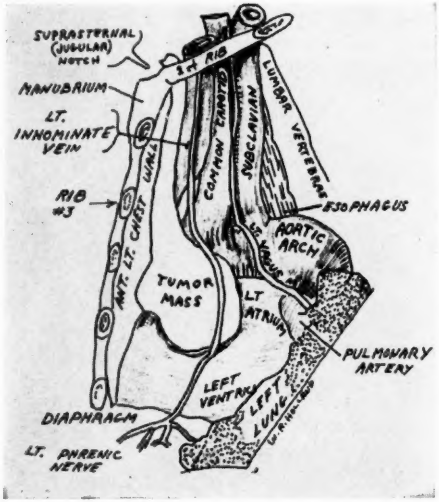


Fig. 2.—(Case 1.) Diagrammatic sketch of the thymic cyst at left thoracotomy and its relationship to mediastinal structures.

Pathologic Examination.—The specimen consisted of a cystic mass which was roughly triangular in shape and measured 10 by 6 by 3 cm. in size. The outer surface was slightly lobulated, fairly smooth, and brown (Fig. 3, *A*). The cyst contained 15 c.c. of chocolate-brown thick fluid and revealed a shaggy, grayish-brown, trabeculated inner surface (Fig. 3, *B*). Several flattened blood clots were adherent to the inner lining. Closer examination showed numerous smaller cysts within the wall of the major cyst. The thickness of the latter varied from 0.3 to 1.3 cm. Although the specimen was multicystic, the smaller cysts probably communicated with the central large cyst inasmuch as the smaller cystic spaces were found to be devoid of fluid after the main cyst was drained.

Microscopically, the cyst and fibrous trabeculations were lined by low cuboidal or pavement epithelium (Fig. 4). Medullary and cortical thymic tissue elements were immediately adjacent to this epithelial lining or separated from it by collagenous fibrous connective tissue of varying thickness. Hassall's corpuscles were present and most of them were calcified. Clotted blood and irregular or spherical masses of thymic medullary tissue were attached to the cyst lining or lay free in the lumen. There were scattered foci of cholesterol clefts surrounded by foreign body giant cells.

CASE 2.—L. S., a 26-year-old white man, was found to have an abnormal anterior mediastinal shadow on a routine chest roentgenogram (Fig. 5). There was a convex density which projected to the right of the midline in the region of the ascending aorta, extending from the level of the sixth to the eighth thoracic vertebrae. The mass was not clearly visible on the lateral film. The patient was asymptomatic. There was no history suggestive of either congenital or acquired syphilis. Physical examination and laboratory studies were within normal limits. The Kahn test was negative.

On Dec. 30, 1952, a right thoracotomy was performed through a posterolateral incision. A free pleural space was encountered. The tumor mass, which was roughly egg shaped, meas-

A.

B.

Fig. 3.—(Case 1.) Photograph of thymic cyst. *A*, Outer surface. *B*, Cut surface. Note the trabeculation of the inner surface.

ured about 6 cm. in the long or vertical axis and 4 cm. in the short or transverse diameter. It was situated anterior to the superior vena cava and in the anteroposterior direction extended from that point to the retrosternal area. Its long axis was vertical. Inferiorly it rested upon the base of the heart and superiorly it extended to a point near the thoracic inlet (Fig. 6).

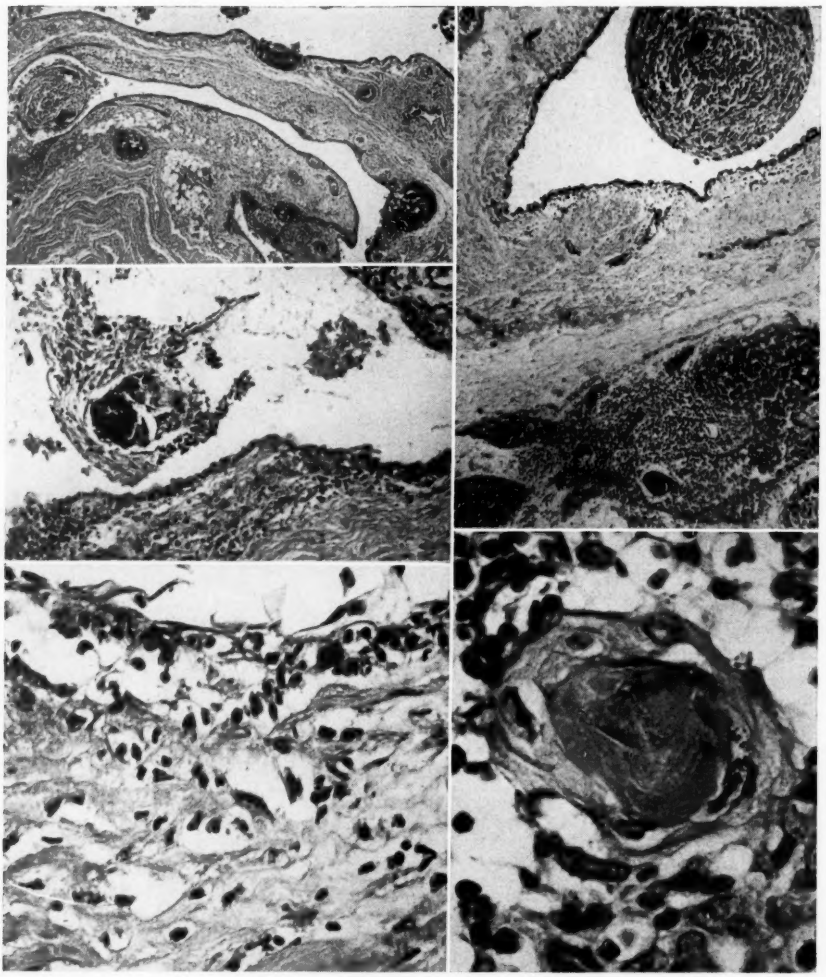


Fig. 4.—(Case 1.) *A*, Photomicrograph showing a fibrous trabecula covered by low cuboidal epithelium (×28). *B*, Cyst containing a sequestrum of medullary tissue including a calcified Hassall's corpuscle (×70). *C*, Higher magnification of cyst lining showing desquamating pavement cells and cuboidal cells covering hyalinized fibrous connective tissue (×300). *D*, Ramification of the cyst separated from thymic cortical substance (lower one-third) by a broad band of fibrous connective tissue (×50). *E*, High magnification of a Hassall's corpuscle (×530).

The mass was largely cystic except for a small solid portion at the inferior pole. The overlying pleura was not adherent to the tumor and stripped from it with ease. At no point was it firmly adherent to the contiguous structures. The lesion appeared well encapsulated and was shelled out and removed without difficulty.

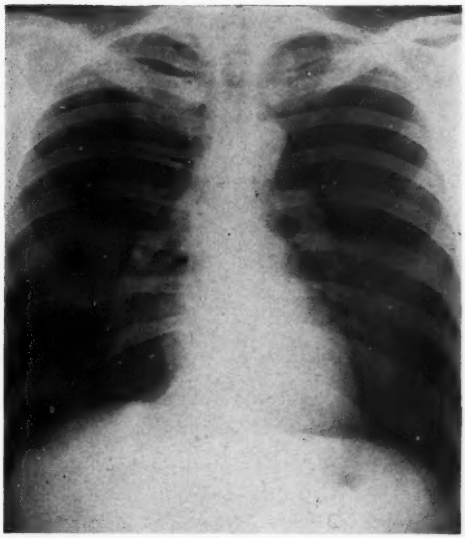


Fig. 5.—(Case 2.) Posteroanterior roentgenogram demonstrating the mediastinal mass.

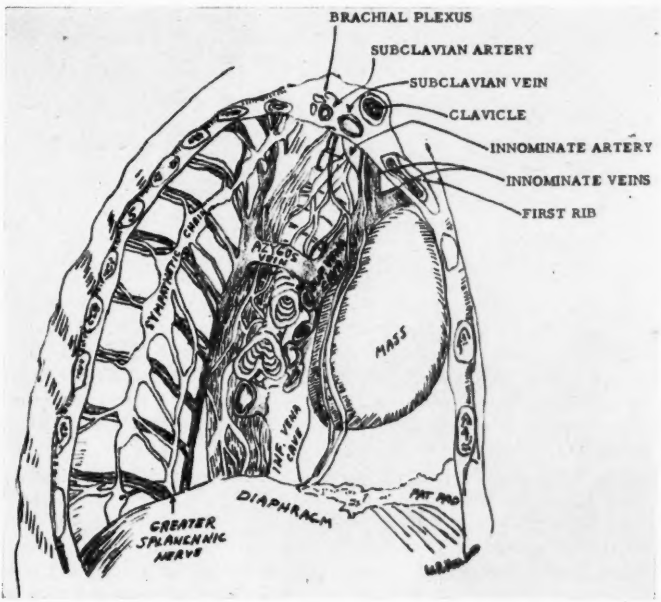
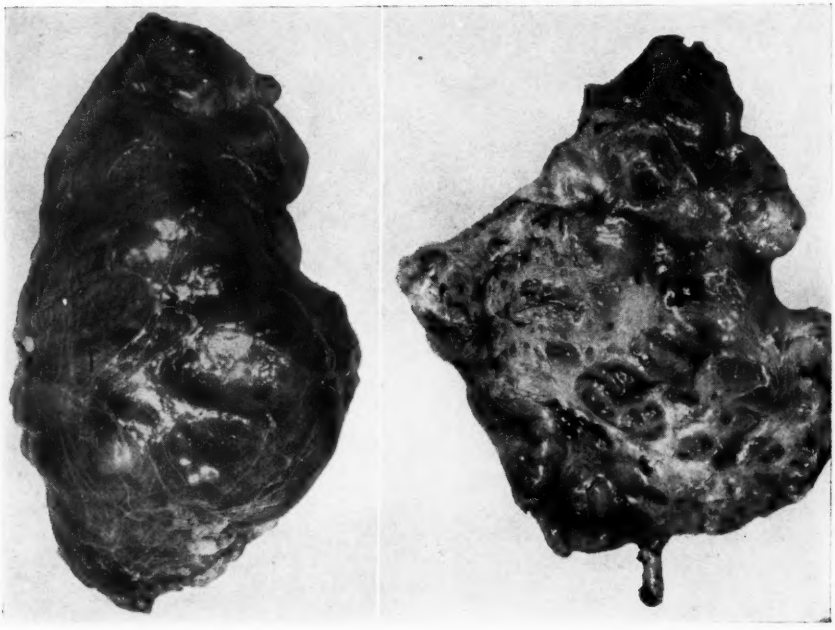


Fig. 6.—(Case 2.) Diagrammatic sketch of the thymic cyst at right thoracotomy and its relationship to mediastinal structures.

Pathologic Examination.—The specimen consisted of an oval-shaped cystic mass which measured 7.0 by 3.5 by 2.5 cm. in size. The outer surface was smooth, lobulated, and reddish-brown in color (Fig. 7, *A*). At the inferior pole the consistency was firmer, suggesting solid

tissue. The cyst contained watery, amber-colored fluid. The cyst wall varied in thickness from several millimeters in the upper portion to several centimeters at the lower pole. Smaller cysts were found within the solid portions and seemed to drain into the large central cavity. The inner surface of the cyst was shaggy and grayish-red, with numerous trabeculations (Fig. 7, *B*).



A. *B.*

Fig. 7.—(Case 2.) Photograph of thymic cyst. *A*, Outer surface. *B*, Cut surface.

Microscopically, the large cyst and its communicating spaces were lined by low cuboidal epithelium or pavement cells. Remnants of medullary and cortical thymic tissue were recognizable in the wall, within the intracystic papillary projections, and in the fibrous trabeculae. Loose, irregularly shaped, or spherical bodies were found within the lumen. They were composed of reticulum cells admixed with thymocytes and inflammatory cells and appeared to represent sequestra of medullary tissue. A few small cystic spaces contained numerous cholesterol crystals.

Discussion of the Histologic Findings.—Examination of multiple sections demonstrated probable stages in the development of the cysts (Fig. 10, *A*). The smallest were situated in the center of a thymic lobule and their linings consisted of medullary reticulum (Fig. 10, *B* and *C*). As the cysts grew larger two changes became apparent. The medullary tissue was compressed and finally disappeared, except for a thin layer of compressed reticulum cells which now appeared as pavement cells. The normally inconspicuous Strandberg connective tissue layer, which forms the corticomedullary boundary, underwent marked thickening by collagenous connective tissue (Fig. 10, *C*). The end result was a three-layered cyst wall consisting of an innermost thin layer of pavement cells, a broad connective tissue layer and a layer of compressed thymic cortical tissue.

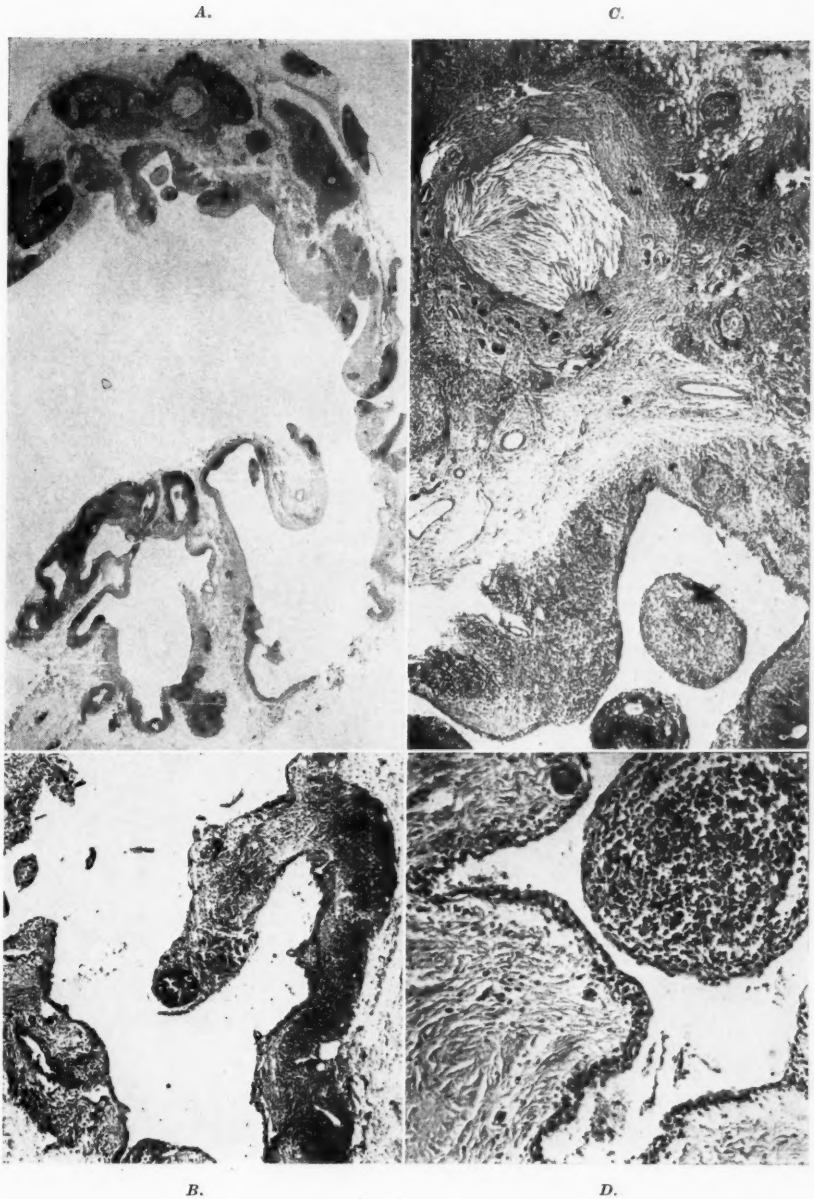


Fig. 8.—(Case 2.) *A*, Photomicrograph showing the cyst with several communicating spaces. Note the lymphoid masses of thymic cortex in the wall. (×6.) *B*, Cyst lined by pavement cells surrounded by thymic cortex. Note the papillary projection containing a Hassall's body in its tip. *C*, Cystic space containing loose spherical bodies (lower portion). A small cyst containing cholesterol clefts is seen in the upper portion. (×28.) *D*, Irregular cyst wall lined by pavement and cuboidal cells and showing a small calcified body beneath the epithelial lining (upper portion of photograph). The loose spherical body is composed of an admixture of reticulum cells and thymocytes.

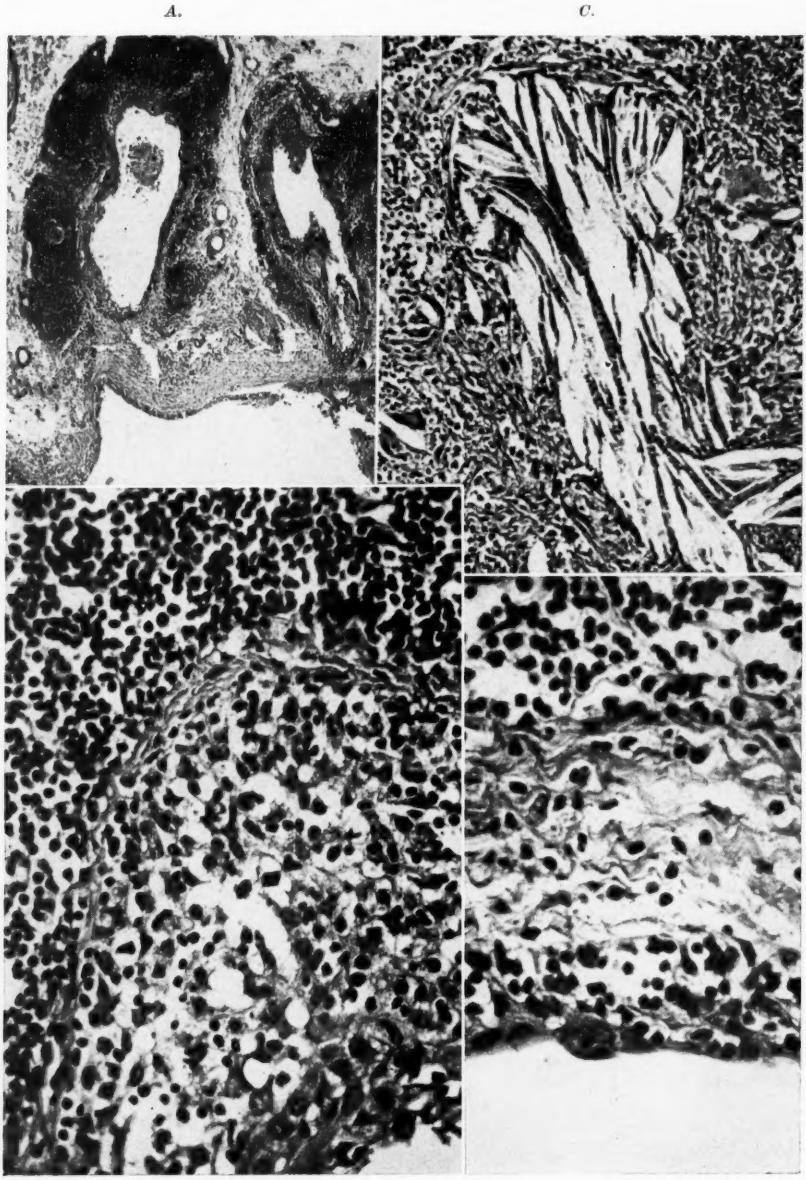


Fig. 9.—(Case 2.) *A*, Three cysts within a thick portion of the wall. The cyst in the upper left is lined by a narrow rim of medullary tissue and a broader zone of cortex. The large cyst in the lower portion of the photograph has lost its reticulum cell and lymphocytic elements. (×28.) *B*, Higher magnification of the cyst in the upper left portion of Fig. 8, *A*. Note the separation of the thymic cortex with its abundant thymocytes from the less cellular medulla by the connective tissue layer of Strandberg. Lining pavement cells are seen in the lower right-hand corner. (×300.) *C*, A focus of cholesterol spaces surrounded by scattered foreign body giant cells. (×390.) *D*, Higher magnification of the cyst seen in the lower portion of Fig. 8, *A*. Four distinct layers from top to bottom consist of (1) thymocytes of cortex, (2) collagenous connective tissue, (3) remnants of medulla, and (4) thin layer of pavement cells lining the lumen. (×300.)

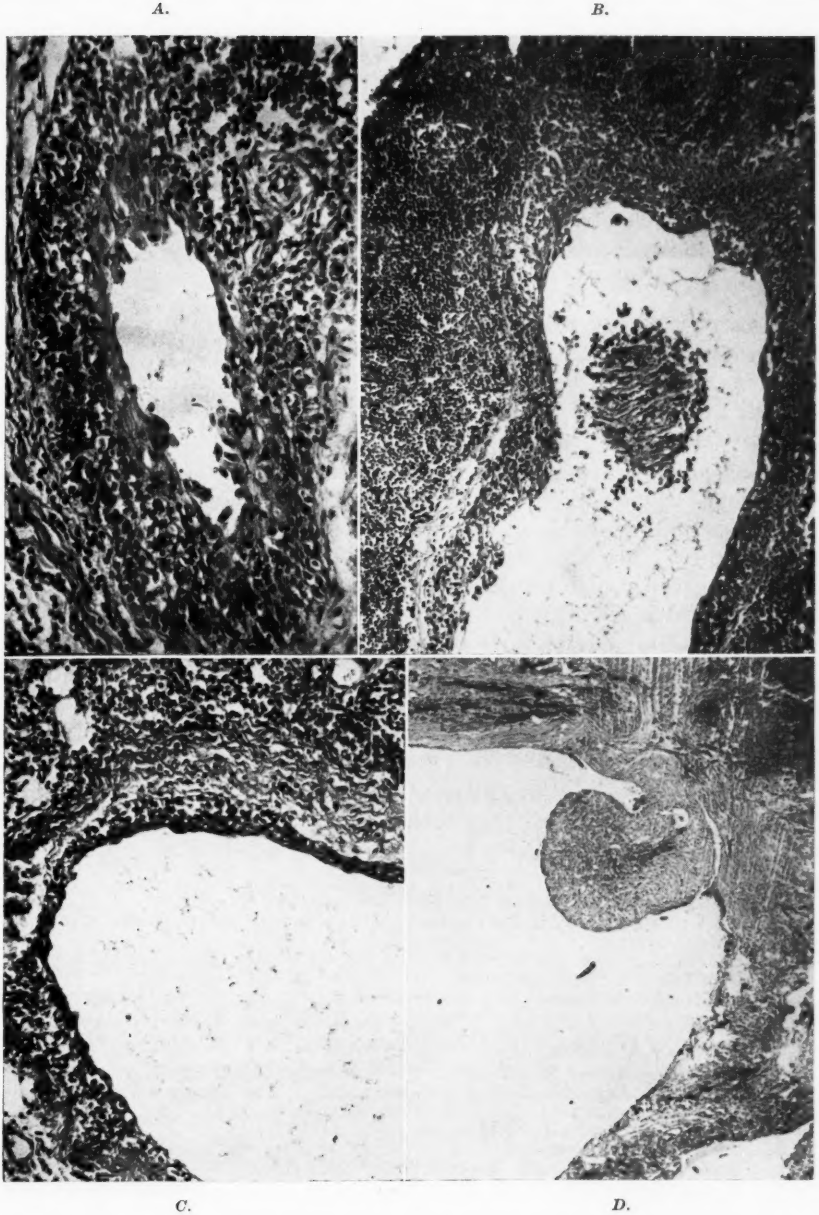


Fig. 10.—Stages in cyst development. *A*, Earliest stage with small cyst within a thymic nodule (×140). *B*, Large cyst with persistent medullary and cortical layers (×70). *C*, Medullary layer almost completely lost. Marked fibrous thickening of Strandberg layer separating cortex and medulla (×115). *D*, Final stage with only collagenous connective tissue lining the cyst (×28).

TREATMENT

The preoperative diagnosis of a thymic cyst is presumptive. Any undiagnosed cervical or anterior mediastinal mass may represent such a lesion. The presence of a mass that could be a cyst of the thymus is the indication for its removal. The cyst should be completely excised. This is usually accomplished either through a cervical or thoracic approach, dependent upon the location of the mass. At times a combined approach may be required in order to accomplish complete removal of a thymic cyst which lies partly in the neck and partly in the mediastinum.

SUMMARY

Two thymic cysts, which presented as anterior mediastinal masses on chest roentgenograms and were diagnosed by histologic examination following excision at thoracotomy, have been reported. Eight additional thymic cysts excised through a cervical incision or at thoracotomy, or both, have been tabulated from the literature. The literature pertinent to this subject has been reviewed.

A simple classification of thymic cysts has been proposed. (The thymic cysts which we have reported and collected were noninflammatory, nonneoplastic, and were probably of congenital origin.) Thymic cysts become clinically manifest when fluid accumulation or hemorrhage occurs into a thymic duct or thymopharyngeal duct remnant. The cause of this phenomenon has not been established.

Thymic cysts present in the neck or anterior mediastinum and usually give rise to no symptoms. They may be unilocular or multilocular. The lining epithelium is characteristically cuboidal or pavement-type epithelium. These cysts commonly contain fluid with fresh or old blood elements and cellular debris within their cavities.

An accurate preoperative diagnosis of these lesions is usually impossible. The treatment of choice is total excision.

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ADDENDUM

Two additional thymic cysts have been resected since submission of manuscript.

Case 3.—W. L. R., a 25-year-old white man, who complained of chest pain on deep inspiration, was found on chest roentgenogram to have a large anterior mediastinal mass projecting into the left chest, and occupying the thoracic inlet. This was removed at left thoracotomy. The specimen measured 12 by 8.3 by 5.5 cm., and was rough and shaggy on the outer surface. On section it appeared as a trabeculated pseudolobulated cyst, with smooth lining, and containing blood clots. The lining was of squamous epithelium. Hassall corpuscles were present in the wall of the cyst.

Case 4.—C. W. N., a 33-year-old white man, was seized with sharp stabbing pain in the throat and left neck. Chest roentgenogram revealed a large anterior mediastinal mass projecting into the right chest. This was removed at right thoracotomy. The specimen measured 13 cm. in greatest diameter, with a fibrous shaggy outer surface. On sectioning it contained large blood clots and liquid bloody material within the prominent trabeculations. The lining was of squamous epithelium. A parathyroid gland was found on microscopic examination in contact with the cyst wall.

THE RELATIONSHIP BETWEEN MEDIASTINAL LIPOMAS AND THE THYMUS

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NEW YORK, N. Y.

A PATIENT was recently found at operation to have a mediastinal tumor containing thymic and fatty tissue. This unusual tumor aroused our interest because the possibility suggested itself that mediastinal lipomas may originate in the thymus, as a result of the infiltration of the organ with fat during its involution. A review of the literature of so-called lipothymomas (the tumor encountered in our patient was representative of this type) and lipomas of the mediastinum gave support for this hypothesis. It was noted that, with few exceptions, intrathoracic lipomas are located in the anterior mediastinum in front of the heart and great vessels. Many of the large lipomas, exceeding the bounds of the mediastinal compartment, were connected by a pedicle or a projection of tissue with the thymic area. Particularly significant was the finding of thymic tissue in some of these lipomas. It can be assumed, therefore, that mediastinal lipomas may be related to the thymus.

CASE REPORT

E. C., a 19-year-old woman from Puerto Rico, was admitted to the Thoracic Surgical Service of Morrisania City Hospital on July 23, 1952, complaining of exertional chest pain of six months' duration. The pain was sharp, nonradiating, and localized to the second and third intercostal spaces to the right of the sternum. Five years previously a chest x-ray film, taken after a seizure of pneumonia, had been interpreted as showing enlargement of the heart. The patient was advised to consult a cardiologist, which she did not do.

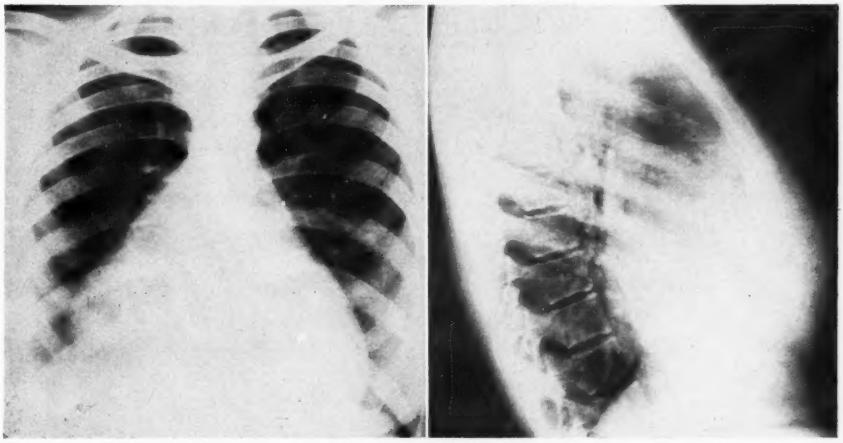
On April 10, 1952, she was admitted in labor to the Obstetrical Service of the Morrisania City Hospital. A chest roentgenogram at this time revealed a large mass in the anterior mediastinum to the right of the heart (Fig. 1). It was triangular in shape with the base resting on the diaphragm, the apex blending with the superior mediastinum. Noteworthy was the decreased density of the peripheral portion of the tumor in comparison to the central area. Following an uneventful delivery, the patient was discharged and advised to return after convalescence. On readmission to the hospital two months later the patient stated that shortness of breath had increased in severity.

Physical examination revealed a well-developed woman not in respiratory distress. Except for dullness and diminished breath sounds over the right chest anteriorly, no other physical abnormalities were found. Routine laboratory studies were within normal limits. Chest x-ray examinations revealed findings similar to those noted upon the first admission. A bronchoscopy was negative. Angiocardiographic studies* showed the mass to be unrelated to the heart and great vessels. From the radiographic appearance of the tumor, a mediastinal lipoma was considered the most likely possibility.

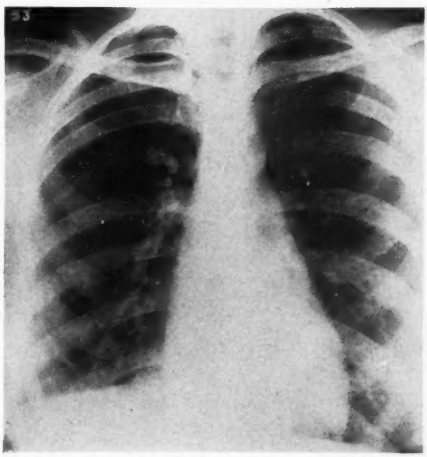
On Sept. 24, 1952, the mediastinum was exposed through a right posterolateral incision. A large, well-encapsulated, lobulated mass was found in the anterior mediastinum adjacent to the heart. The mass was conical in shape with the base on the diaphragm and apex extending as a pedicle into the superior mediastinum. The tumor had the gross appearance of a lipoma. The mass was covered by mediastinal pleura and was easily mobilized from the

From the Thoracic Surgical Service, Morrisania City Hospital.
Received for publication Oct. 26, 1953.
*Done by Dr. W. Rosenbluth.

diaphragm and pericardium. At the apex the pedicle could be traced anteriorly and medially to the superior vena cava into the thymic region behind the sternum. A few blood vessels within the pedicle arborized over the surface of the tumor beneath the pleura. The pedicle



A. *B.*

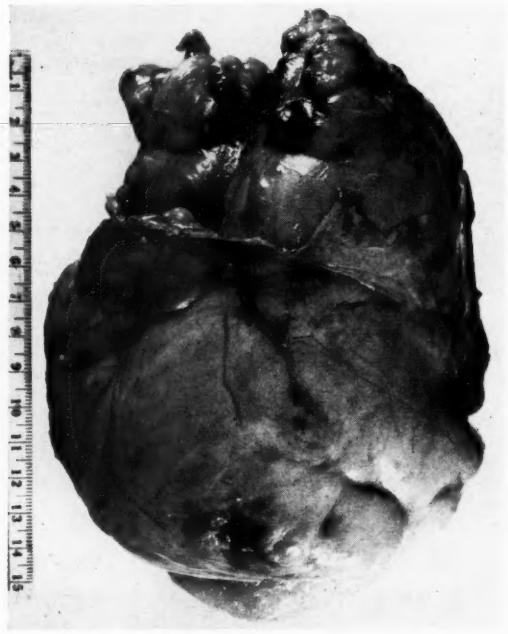


C.

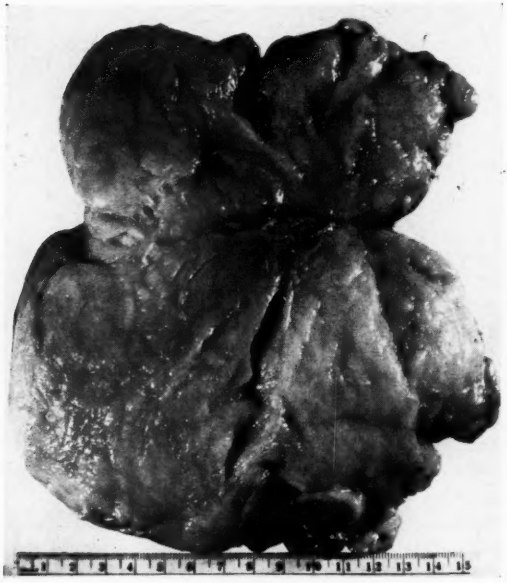
Fig. 1.—A 19-year-old woman with symptoms of dyspnea and chest pain. Diagnosis of an enlarged heart at the age of 14 years. *A*, Anteroposterior view (March 14, 1951) shows a large triangular shadow blending with the cardiac silhouette. Note double density of periphery of mass. *B*, Right lateral view reveals the mass to be in the anterior mediastinum. *C*, Roentgenogram of chest (April 10, 1953) seven months after excision of large thymic tumor of anterior mediastinum.

was easily divided between clamps and the mass delivered from the pleural cavity. The patient made an uneventful recovery and was discharged on the fourteenth hospital day. She has been followed in the clinic for the past twelve months and is well (Fig. 1, *C*).

Grossly, the tumor was an oval, lobulated mass, measuring 20 by 12 by 10 cm. and weighing 750 grams (Fig. 2). It was covered by a thin layer of pleura beneath which a few



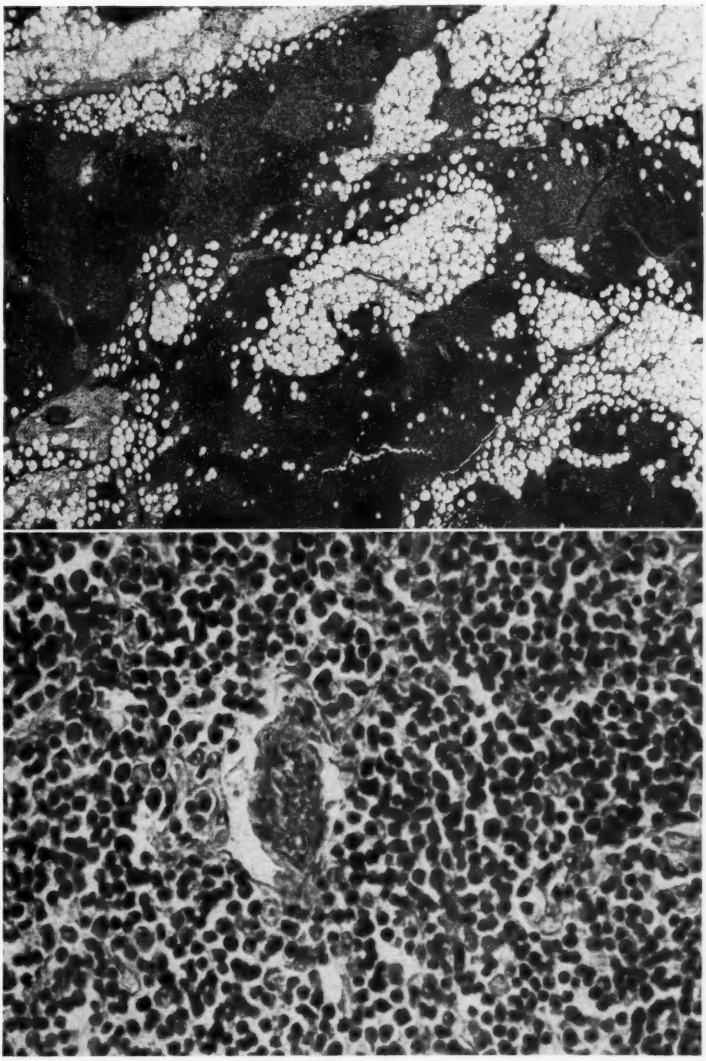
A.



B.

Fig. 2.—Surgical specimen. Lobulated fatty tumor, weighing 750 grams, covered by mediastinal pleura. Upper pole was attached to the thymic region. Cross section showed the tumor to consist of fatty tissue with numerous dense gray areas.

A.



B.

Fig. 3.—Microscopic findings are those of an admixture of thymic and fatty tissue distributed in an irregular manner throughout the tumor. The thymic portion contains both cortical and medullary elements. A Hassall corpuscle can be seen in the high-power view. *A* (×32). *B* (×530).

blood vessels were visible. On section the tumor was found to consist of fatty tissue with numerous denser grayish areas. Microscopic examination revealed a mixture of thymic and fatty tissue (Fig. 3). Both elements were distributed in an irregular manner throughout the tumor mass. The fatty tissue appeared normal throughout. The thymic element was arranged in the lobular pattern strongly suggesting normal thymic tissue with cortical and medullary zones. The cortical portions of these lobules consisted almost entirely of mature adult lymphocytes. Distinct Hassall corpuscles were found sparsely scattered in the medullary portions of these lobules. The corpuscles consisted of irregularly ovoid cores of amorphous basophilic material surrounded by one or two concentric rings of flattened squamoid cells with finely granular deeply eosinophilic cytoplasm and large ovoid nuclei. There was no evidence of malignancy.

DISCUSSION

Benign mediastinal tumors consisting of an admixture of thymic and adipose tissue are rare. Only three such cases have been described previously. Andrus and Foot[1] published the first such case in 1937. The patient was a 13-year-old boy who was found to have a large mediastinal tumor which was producing dyspnea and cough. At operation an enormous tumor weighing 2,235 grams was successfully removed. The tumor was found to contain thymic tissue and fat. The second case was published by Bigelow and Ehler in 1952.[2] The patient was a 10-year-old girl who had cough and bloody sputum. A similar thymic tumor was removed successfully. The patient was followed for four years without evidence of recurrence. A third case, described by Gross[3] in his recent book, was that of a thymoma removed from a 14-year-old boy, who was found to have a mediastinal mass following routine chest film examination. It was thought to be a lipoma before operation. At operation a tumor was found in the anterior mediastinum extending down from the left lobe of the thymus. It was composed of lymphoid elements and large amounts of fatty tissue. The child was well six months after operation. Bariety and Coury[4] quote a possible fourth case of lipothymoma treated by Houdard. Unfortunately Houdard's report was not available for study.

The cases cited have certain features in common. They were all found in children between the ages of 10 and 15 years. We are assuming that the mass in our patient was present when a so-called enlarged heart was found in the chest x-ray examination at the age of 14 years. Such tumors may simulate cardiac enlargement as shown in a case report by Gottlieb, Baer, and Jordan.[22] In their patient, a 30-year-old white man, a mediastinal lipoma, attached to the pericardium, was diagnosed originally as cardiac enlargement. With the exception of the patient reported by Gross, the others had symptoms referable to pressure of the tumor; none had symptoms of myasthenia gravis nor was there evidence of malignancy. Bigelow and Ehler coined the term, "lipothymoma," which is descriptive of the disease and apparently acceptable for this type of tumor. The term, lipothymoma, was also used by Houdard. Whether or not these tumors are true thymomas or hyperplasia of both the thymic and fatty tissue is difficult to say.

These rare tumors, occurring apparently in children only, present a number of interesting problems as to their pathogenesis and possible relationship to mediastinal lipomas. The thymus originates as paired structures from the

third and fourth gill arches in the same general region as those of the parathyroid gland. The structures descend laterally in the neck and, after meeting in the midline, enter the anterior part of the superior mediastinum behind the sternum. Thymic rests may occur along the course of migration into the mediastinum. The subsequent development of the thymus is one of enlargement during childhood reaching a maximum in the late teens followed by a slow involution with replacement by adipose tissue in which remnants of thymic tissue may be present. Thus, it is conceivable that tumors of the thymus during this period of growth and involution may consist of both thymic and fatty components. With involution of the thymic element, the lipomatous portion may continue to grow. This idea is supported by the fact that lipothymomas have not been described in the older age groups. It is also possible that a lipoma can originate from thymic fat after involution had occurred. In either case some anatomic relationship of lipomas with the thymic area should have been noted. With this view in mind, an analysis was made of the recorded cases of mediastinal lipomas.

Of the fifty-eight lipomas recently analyzed by Keeley and his co-workers,[21] forty-two were entirely in the anterior mediastinum; six, in the cervicomediastinal region. Ten were dumbbell shaped, composed of an intra- and extrathoracic component connected by an isthmus of tissue through an opening in an intercostal space or, more rarely, in the sternum. Some doubt has been raised whether this last group should be included with the intrathoracic lipomas. The fact, nevertheless, remains that in all of these cases some anatomic relationship to the anterior mediastinum was present. Lipomas in the posterior and middle mediastinum are rare.[26]

An analysis was made of the gross and microscopic findings of the reported cases with reference to points of attachment and possible thymic tissue remnants. In some of the cases the description of the operation or necropsy findings was too brief to permit analysis. A relationship with the thymic region was recorded in twelve of the larger tumors as may be gathered from such statements as "a pedicle extending to the suprasternal notch," "a pedicle from the arch of the aorta," "projections in the thymic area," or "projections in the superior mediastinum."[4, 6-16] An unpublished case of a mediastinal lipoma with a pedicle attachment to the thymic area which was removed surgically was described to us by Dr. J. M. Chamberlain.[13]

Thymic remnants were mentioned in three cases of mediastinal lipomas. Such tissue was present in the pedicle in a case reported by Beatson.[7] Similar tissue was found in the center of the lipoma in the cases reported by Bariety and Coury[4] and Schaner and Hodge.[6] It is conceivable that thymic remnants might have been demonstrated in other cases if sufficient blocks of tissue were taken, particularly from the center of the tumor.

The mode of migration of the thymus helps to explain the limitation of intrathoracic lipomas to the cervicomediastinal and mediastinal region. In this respect Lemon's[9] case is particularly interesting. The patient had a small lipoma in the supraclavicular area in addition to a large one in the anterior mediastinum which was attached to the trachea, aorta, and bronchi. An example of a

cervicomediastinal lipoma which was removed through a cervical incision is one reported by Graham and Wiese.[18] Whether such lipomas result from thymic rests is an interesting point of conjecture.

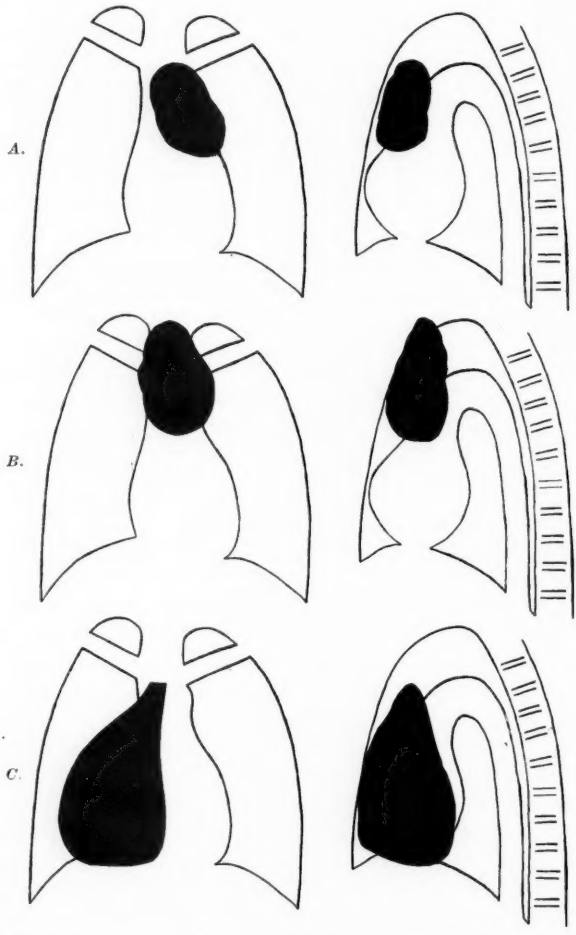


Fig. 4.—Diagrammatic presentation of the anatomic locations of intrathoracic lipomas. *A*, Anterior mediastinal. *B*, Cervicomediastinal. *C*, Anterior mediastinal showing downward projection of the large tumors. The resulting shape of the mass is probably influenced by its point of fixation in the thymic region and its semisolid consistency at body temperature as it comes to rest on the diaphragm.

It is thus possible to reconstruct the evolution of the larger lipomas as follows: A lipoma or lipothymoma may arise from the thymus and may remain localized to the thymic area or along its course of migration into the chest (Fig. 4, *A*) if its period of growth is short or if it is detected early. Such tumors may, therefore, project upward into the neck, forming a tumor of the cervicomedias-

tinal type (4, *B*). In view of the semisolid consistency of fat at body temperature, the further growth of the tumor influenced by gravity is downward. The tumor, descending into the anterior mediastinum in front of the heart, soon rests on the diaphragm. It there begins to spread laterally, and may project into both pleural cavities or remain localized to one side. Because it maintains a fixed relation to its point of origin, it assumes a flasklike contour, a feature characteristic of such tumors before they become very large (Fig. 4, *C*). This "Erlenmeyer flask" appearance of lipomas, due to their attachment at the superior pole, is clearly shown in the specimen removed by Smart and Thompson.[16]

If the tumor is discovered early in life both thymus and fatty elements may be found. On the other hand, if involution has taken place, fatty tissue predominates. Lipomas and lipothymomas of comparable size have, therefore, a similar radiographic appearance. Considering the age group in which the lipothymomas are found, it is conceivable that with involution of the thymic element, the fatty element may persist as a lipoma. Because of the slow growth of the tumors, symptoms are absent until later in life when the mass has become sufficiently large to interfere with the functions of the heart and lungs.

The relationship of the thymus to mediastinal lipomas bears resemblance to other tumors found in the same region which are also considered to originate from the same anlage as the thymus; namely, dermoids and teratomas. Hedblom divided these tumors into those of the anterior mediastinum, and of the retrosternal and suprasternal areas.[29] Hourglass and presternal varieties have also been described. Rusby[28] describes dermoids as midline tumors which often have a pedicle stretching from the tumor to the center of the chest. He also mentions that the most reasonable explanation of the origin of these tumors is from abnormalities of the third and fourth gill arches, an area where development assumes a great degree of complexity due to the relationship of the various germinal layers. Schlumberger,[27] in a study of sixteen teratomas of the anterior mediastinum, held that such tumors probably arise from tissue dislocation in the anlage of the thymus. It is more than a coincidence that two tumors which are considered related to the thymus should have a comparable topographic location.

SUMMARY

A tumor of the thymus consisting of an admixture of thymic and adipose tissue is discussed. Such tumors, found in children only, have been designated as lipothymomas. Certain similarities between lipomas and lipothymomas are noted which raise the possibility of a developmental relationship between mediastinal lipomas and the thymus. A review of the embryogenesis of the thymus and the reported cases of lipomas and lipothymomas favor such a relationship.

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AN EVALUATION OF EXTRAPLEURAL PNEUMONOLYSIS WITH FILLING WITH PLASTIC BALLS

Analytic Studies on Sixty-Three Patients From Whom Plastic Balls Were Removed

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INTRODUCTION

SINCE Tuffier first performed "extraparietal stripping" in 1891, the operation has been used with modifications by thoracic surgeons all over the world. To maintain the collapse effected by extrapleural separation of the parietal pleura from the chest wall, a multitude of substances have been used, including air, fat, muscle, paraffin, oil, gauze, Cellophane, rubber, fiberglass wool, and many other substances. For a time paraffin was widely used, but because of complications, especially lung perforation, this method also gradually fell into disuse.

In 1948, Wilson[1] reported extrapleural pneumonolysis with Lucite plombage. Subsequently Trent and his associates,[2] in 1948, reported experiences with fifty-one cases in which this method was used. Independently from Wilson, in Japan, in 1947, Nagaishi, Tsuji, and Minoguchi[3, 4] found that Lucite in the form of spheres was inert in the extrapleural space and maintained a satisfactory collapse of the lung; they reported good results in 126 cases in 1948. However, in spite of the early good results, Wilson, in America, emphasized the experimental nature of the operation and warned against excessive optimism. Trent discarded this operation in January, 1947, when late complications began to occur; he reported his experiences with this operation in 1948.

In Japan, the simplicity of the operation and the immediate good results appealed to both the patients and the surgeons, and many patients refused to have any but the "ball" operation; the indications for this operation were broadened to include those which would usually suggest thoracoplasty as the operation of choice. The operation of the plombage type was widely used throughout Japan.

Since 1949, many reports have been found in the literature in Japan. At first many encouraged use of this operation. When adequate follow-up studies were made, Sawazaki[5] reported results with 1,064 patients, who were operated

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TABLE I

CASE NO.*	INDICATION FOR PLOMBAGE	INTERVAL BETWEEN FILLING AND REMOVAL	REASON FOR REMOVAL	MEMBRANE BETWEEN BALLS	FLUID ACCUMULATION IN BALLS		LUNG PERFORATION	BLOOD LOSS DURING OPERATION (GM./KG.)	RESULTS
					PRESENCE§	CHARACTER			
1	Bad (Lobité supérieure)	5 mo.	Empyema	Liquefied	–		+ 1		Sinus formation; at present sinus cured by reoperation
2	Good	6 mo.	Empyema	Liquefied	–		–		Sinus formation; at present sinus cured by reoperation
3	Bad (giant cavity)	10 mo.	Positive sputum†	Hard	–		–	12.0	Good
4	Roentgenogram before plombage not available	10 mo.	Empyema	Liquefied	–		–		Good
5	Bad (giant cavity)	5 mo.	Positive sputum†	Hard	–		–	6.5	Temporary sinus formation, due to gauze which remained in wound at operation
6	Good	1 yr.	Positive sputum†	Hard	–		–	10.7	Good
7	Bad	11 mo.	Positive sputum†	Slightly fragile	–		–	10.4	Good
8	Good	9 mo.	Fluid in balls	Slightly hard	+ *1*	Serous, turbid	–	4.7	Good
9	Bad	1 yr., 5 mo.	Positive sputum†	Slightly fragile	+ *1*	Pus	–	5.8	Good
10	Good	6 mo.	Positive sputum‡	Hard	+ *1*	Pus	–	Severe	Operative death
(11)	Bad	7 mo.	Fluid in balls	Fragile	+ *1 br.* – *br.*	Pus	–	8.1	Sinus formation, cavity entered
12	Bad (Lobité supérieure)	2 yr., 5 mo.	Lung perforation	Liquefied			+ 6	Severe	Operative death
(13)	Bad (Lobité supérieure)	1 yr., 11 mo.	Empyema	Liquefied	– *br.*		+ 1	14.3	Good
14	Good	1 yr., 9 mo.	Positive sputum‡	Fragile	+ *4*	Serous, brownish	Necrotic, 2 punctured	11.0	Good
15	Bad	2 yr.	Positive sputum‡	Fragile	–		–	8.2	Good

Footnotes on p. 506.

TABLE I—CONT'D

CASE NO.*	INDICATION FOR PLOMBAGE	INTERVAL BETWEEN FILLING AND REMOVAL	REASON FOR REMOVAL	MEMBRANE BETWEEN BALLS	FLUID ACCUMULATION IN BALLS		LUNG PERFORATION	BLOOD LOSS DURING OPERATION (GM./KG.)	RESULTS
					PRESENCE§	CHARACTER			
16	Bad	2 yr., 10 mo.	Empyema	Liquefied	+ *1 br.*	Pus	–	13.8	Good
(17)	Good	2 yr., 6 mo.	Wish	Fragile	–		–	12.8	Good
18	Good	2 yr. 7 mo.	Fluid in balls	Fragile	+ *1*	Serous	–	5.8	Good
19	Bad	2 yr.	Fluid in balls	Slightly fragile	+ *2*	Bloody	–	14.6	Good
20	Bad	1 yr., 3 mo.	Wish	Fragile	–		–	7.1	Sinus formation
21	Good	3 yr., 4 mo.	Fluid in balls	Fragile	+ *2*	Pus	–	14.9	Good
(22)	Bad	2 yr., 2 mo.	Lung perforation	Slightly hard	+ *1 br.*	Pus	6	10.5	Wound separation
23	Good	2 yr., 6 mo.	Wish	Slightly hard	–		–	12.3	Good
24	Good	2 yr., 6 mo.	Wish	Hard	– *br.*		–	11.6	Wound separation
25	Bad	2 yr., 4 mo.	Empyema	Liquefied	–		–	7.4	Good
26	Good	2 yr., 6 mo.	Positive sputum‡	Fragile	–		–	13.2	Good
27	Good	2 yr., 2 mo.	Positive sputum‡	Fragile	+ *1*	Pus	+ 1 autopsy	Severe	Operative death
28	Good	3 yr., 1 mo.	Fluid in balls	Liquefied	+ *1*	Pus	–	8.4	Good
29	Bad	2 yr., 6 mo.	Positive sputum‡	Fragile	–		–	15.1	Good
30	Good	2 yr.	Wish		+ *2*	Serous, black	–	10.7	Good
31	**	3 yr., 3 mo.	Fluid in balls		+ *3 br.*	Pus	–		
32	Good	2 yr., 8 mo.	Positive sputum‡		–		–	6.1	Good
33	Good	1 yr., 6 mo.	Fluid in balls	Fragile, partly hard	+ *2*	Serous	–	10.9	Good
34	Good	3 yr.	Hemoptysis	Fragile	–		–	8.3	Good
35	Bad	2 yr., 3 mo.	Positive sputum‡	Fragile, partly liquefied	+ *1*	Pus	–	11.8	Good
36	Good	1 yr., 9 mo.	Fluid in balls	Fragile	+ *1*	Pus	–	5.5	Good
37	**	1 yr., 10 mo.	Wish	Fragile	–		–	4.2	Good
38	Good	1 yr., 11 mo.	Fluid in balls	Fragile	+ *1*	Serous, brownish	–	9.2	Good
(39)	**	2 yr., 3 mo.	Empyema	Liquefied	–		–	11.7	Good
40	Bad	3 yr., 1 mo.	Fluid in balls	Fragile	+ *2 br.*	Serous, brownish	–	13.3	Good
41	Good	2 yr., 3 mo.	Fluid in balls	Fragile	+ *1*	Serous, brownish	–	14.8	Good
42	Bad	2 yr., 3 mo.	Positive sputum‡	Fragile	–		–	7.4	Good
43	Good	2 yr., 7 mo.	Positive sputum‡	Fragile	–		–	15.3	Sinus formation

Footnotes on p. 506.

TABLE I—CONT'D

CASE NO.*	INDICATION FOR PLOMBAGE	INTERVAL BETWEEN FILLING AND REMOVAL	REASON FOR REMOVAL	MEMBRANE BETWEEN BALLS	FLUID ACCUMULATION IN BALLS		LUNG PERFORATION	BLOOD LOSS DURING OPERATION (GM./KG.)	RESULTS
					PRESENCE§	CHARACTER			
44	Good	3 yr., 1 mo.	Fluid in balls	Fragile	+ *2*	Serous, brownish	–	13.0	Good
45	Good	2 yr., 6 mo.	Fluid in balls	Fragile, partly liquefied	+ *3 br.*	Pus	–	8.2	Wound suppuration
(46)	**	2 yr., 6 mo.	Increased sputum	Slightly hard	*1*	Serous	–	9.0	Good
47	**	2 yr., 8 mo.	Positive sputum‡	Hard	–				Good
48	Good	2 yr., 3 mo.	Fluid in balls	Liquefied	+ *3*	Pus	–	14.1	Good
49	Good	2 yr., 3 mo.	Wish	Slightly hard	–		–	14.1	Good
50	Good	1 yr., 11 mo.	Fluid in balls				–	9.2	Good
51	Good	2 yr., 1 mo.	Fluid in balls	Hard	+ *1*	Serous, fibrin mass	–	18.2	Good
52	Good	3 yr., 1 mo.	Fluid in balls	Partly hard	+ *1*	Pus	–	5.5	Good
53	Good	1 yr., 9 mo.	Fluid in balls	Fragile	+ *1*	Pus	–	6.7	Good
54	Good	2 yr., 9 mo.	Fluid in balls	Hard, partly liquefied	+ *2*	Serous, fibrin mass	–	11.2	Good
55	Good	2 yr., 3 mo.	Fluid in balls	Fragile	– At operation fluid was not found		–	8.7	Good
56	Good	2 yr., 11 mo.	Empyema	Liquefied	+ *2*	Serous	–	14.7	Good
57	Good	3 yr., 6 mo.	Fluid in balls	Fragile	+ *4*	Pus	–	8.4	Good
58	Bad	3 yr., 2 mo.	Fluid in balls	Fragile	+ *3*	Serous, brownish	–	8.6	Good
59	Good	2 yr., 7 mo.	Empyema	Liquefied	+ *8 br.*	Pus	Necrotic, 2 punctured	8.9	
60	Good	2 yr., 3 mo.	Fluid in balls	Fragile	–		–	12.6	
61‖	Bad	2 yr., 5 mo.	Positive sputum†	Fragile	–		+ 1		Sinus formation
62‖	Bad	1 yr., 2 mo.	Positive sputum†	Hard	–		–	10.6	Good
63‖	Bad	2 yr., 9 mo.	Positive sputum‡	Hard	–		–	9.1	Good

*The case numbers which appear in parentheses indicate that the patient was operated on at a hospital other than the National Okayama Sanatorium.

**Roentgenogram before plombage not available.

†Positive sputum in direct smear.

‡Positive sputum in concentrated smear.

§The italicized numbers indicate the number of balls in which fluid was accumulated. *br.* indicates breakage of a ball.

‖Plombage following thoracoplasty in Cases 61, 62, and 63.

upon by March, 1949, at forty national sanatoriums and on whom follow-up study was carried out from five months to two years after the operation (80 per cent after five to eleven months), showed 50 per cent had improved, 32 per cent were unchanged, 7 per cent were unimproved, and 6 per cent were dead. Complications reported from the operation included: twenty-four postoperative cavity perforations (2.2 per cent), four cavity perforations during operation, eight bronchopleural fistulas, thirteen sinus formations, seven tuberculous extrapleural empyemas, and eight nontuberculous empyemas. One year later, he[6] reported that lung perforation increased from 2.2 per cent to 7.5 per cent and empyema from 0.6 per cent to 3.5 per cent in the same series. The incidence of these complications widely varied in each sanatorium. It is generally accepted that the less involvement of the lung there is before operation the fewer the complications that will occur. Nagaishi[7] reported eleven perforations (2.8 per cent) in 393 cases. Sawazaki[8] reported 5.4 per cent in 148 cases at National Hiroshima Sanatorium in 1950. The highest reported incidence of lung perforation was 17.8 per cent in 90 cases in Kamo National Sanatorium.[8]

Today surgeons in sanatoriums are busily occupied in removing these balls. In National Okayama Sanatorium from May, 1948, until March, 1950, ninety-seven patients were operated upon by this method; a total of 106 operations was performed. Up until this time three operative deaths, one death due to hemoptysis with lung perforation, six lung perforations, and six empyemas have occurred, and in fifty-seven cases extrapleural plastic balls were removed for varied reasons. It is evident that the longer the cases are followed up the more the complications become evident. This report is based on an analysis of sixty-three cases, in which extrapleural plastic balls were removed from the patients. We feel that it is important to report our experiences with this reoperation at this time, for little literature is available on operations of this type. The practice of removing the extrapleural plastic balls is spreading and some dangers accompany an operation of this kind.

ANALYSIS ON REMOVAL OF EXTRAPLEURAL PLASTIC BALLS

In total, sixty-three operations were performed on sixty-two patients. Of these, sixty were primary plombage and three were plombage following thoracoplasty in which the cavity had not been closed (Table I). The reasons for the removal of the plastic balls are shown in Table II.

In Table I, in the column headed "Indication for Plombage" the word *good* refers to a patient, who was in good general condition and had a localized lesion

TABLE II. REASON FOR REMOVAL OF PLOMBAGE

REASON	NO. OF PATIENTS
1. Extrapleural empyema	6
2. Lung perforation	2
3. Fluid accumulation in balls	27
4. Positive sputum	18
5. Patient's wish	8
6. Hemoptysis	1
7. Increase of sputum	1

in upper lung field with small cavity situated at a distance from the chest wall or without evident cavity and whose sputum contained scanty tubercle bacilli or no bacilli. *Bad* refers to a patient, who had a large cavity, especially one situated close to the chest wall, or so-called "lobité supérieure" (extensive lesion) and also those patients whose sputum was highly laden with acid-fast bacilli. Complications occurred more frequently in patients who had bad indications. But it is noteworthy that in the course of time, postoperative complications, especially the side-effects of the plastic ball itself, such as fluid accumulation in the balls, markedly increased irrespective of preoperative indications.

OPERATIVE FINDINGS

1. Lung Perforation.—At operation perforation of lung was found in four patients; one was not clinically detected prior to operation. A small, brownish-black necrotic change on the parietal pleura, which was considered as the beginning stage of perforation, was found in two patients.

2. Extrapleural Empyema.—Empyema was found in nine cases, in 3 of which it was not diagnosed preoperatively.

3. Fluid Accumulation in Balls.—Fluid accumulation in the plastic ball was found in thirty-two cases. Seventeen of the thirty-two were suppurative including apparently tuberculous granulation mass; others were serous or slightly turbid and their color varied, some contained a fibrin mass or a granulomatous mass. Broken plastic balls were found in nine patients, and in most fluid was existent. In eleven cases the tubercle bacillus was detected by culture from these. It is noticeable that fluid accumulates more or less in the balls in the course of time, for this suggests that inflammation, mostly tuberculous infection, occurs in the extrapleural space sooner or later.

4. Capsule Between the Balls.—The membrane between the balls was very firm in cases which did not elapse so long after plombage. Therefore, in these early cases, the removal of plastic balls was moderately difficult technically, except in empyema cases. In fact, in one case the balls were so firmly embedded that removal was impossible without endangering the patient's life. In one case the attempt was abandoned after seven balls had been removed and the cavity had been entered; this case developed tuberculous wound infection. But in later cases, in which more than two years had elapsed after plombage, the membrane between the spheres was usually fragile and tuberculous granulation tissue existed in most cases. Therefore, removal of balls was relatively easy in these cases. Of course, in empyema cases the membrane was completely dissolved and the balls were afloat in pus.

OPERATIVE TECHNIQUE

This operation is generally more difficult and blood loss during operation is greater than in an ordinary primary thoracoplasty, for the periosteum is hard and the rib is fragile due to the pressure caused by the balls. In many cases when the apex of the lung had been pushed down by the previous operation it was unnecessary to resect the first rib and transverse process, but it is our

routine procedure to strip the periosteum of the first rib to reduce extrapleural space and thus to prevent the re-expansion of the lung. In one stage it is possible to resect five ribs (from the second to the sixth), for the lung has been moderately collapsed by plombage and the effects on the cardiorespiratory system by rib resection are less than in ordinary primary thoracoplasty. The operative wound was always primarily closed in spite of the existence of lung perforation or empyema.

Of the three patients with lung perforation, one had wound separation and two developed a sinus. But in empyema cases all were cured primarily. Of course, the operation is to be divided and sufficient rib resection is to be performed in the cases in which the lesion of the lung has not been controlled by plombage or in poor-risk cases, that is, rib resection is to be limited to the extent of obliterating the extrapleural space in cases in which the lung lesion has been controlled by plombage.

OPERATIVE RESULTS

There were three operative deaths. These will be briefly described. Postoperative complications are summarized in Table III; it demonstrates that operative complications occur more frequently than in the usual primary thoracoplasty.

TABLE III

POSTOPERATIVE COMPLICATIONS	NO. OF PATIENTS
1. Operative death	3
2. Temporary sinus formation	1
3. Tuberculous sinus formation	3
4. Sinus formation with bronchial fistula	3
5. Wound separation with bronchial fistula	1

CASE REPORTS

CASE 1 (Case 10, Table I).—T. U., a 23-year-old man, was admitted to National Okayama Sanatorium on Sept. 3, 1949. He complained of a small amount of cough and sputum. Concentrated sputum was "loaded" with tubercle bacilli on smear. Direct radiogram of chest revealed slightly dense infiltration in the left upper lung field, and a productive nodular shadow was visible in right upper field. On Nov. 10, 1949, left extrapleural pneumonolysis with plastic balls was performed; on May 15, 1950, right plombage was performed. After plombage, the patient's general condition was good and the complaints almost disappeared, but sputum was occasionally positive by concentration method. Therefore, removal of left extrapleural plombage was attempted on Nov. 21, 1950. All balls were very firmly embedded and removal was very difficult. On removal of the last ball, embedded in anterior apex, massive bleeding abruptly occurred. Because the control of the bleeding was impossible, the wound was primarily closed in three layers after tight gauze packing. Postoperative course was dramatic. Marked swelling of the left chest and neck appeared and the patient complained of tightness and dyspnea. But neither hemoptysis nor bleeding from wound occurred. On the fourteenth postoperative day removal of gauze pack was made. At that time no bleeding occurred, but on the eighteenth postoperative day massive hemorrhage from wound occurred and subsequently 5 similar episodes followed. On the twenty-sixth postoperative day he died from fatal hemorrhage. Autopsy was not allowed. I think the azygos vein was injured upon removal of the last ball.

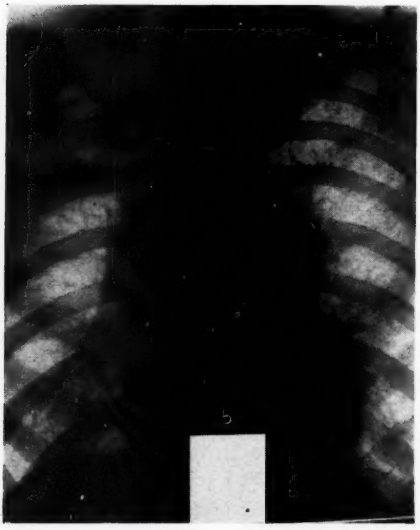
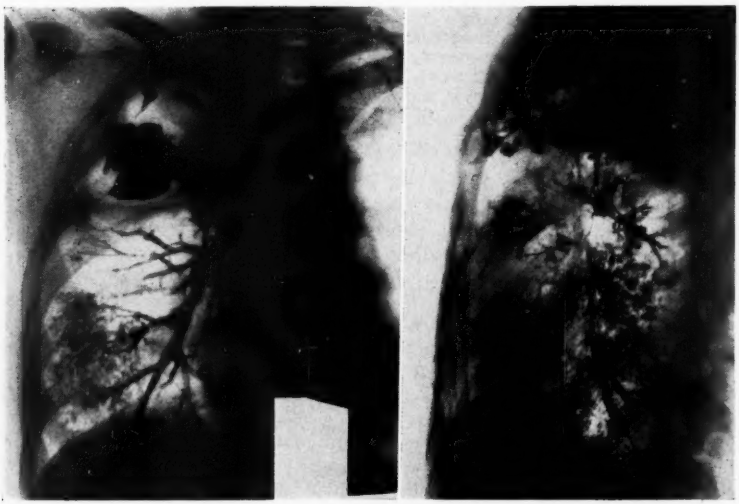


Fig. 1.—(Case 2.) Posteroanterior roentgenogram of the chest showing right extrapleural plombage (Dec. 18, 1950).



A. *B.*

Fig. 2.—*A*, Posteroanterior bronchogram showing lung perforation (Feb. 9, 1951). *B*, Right lateral bronchogram.

CASE 2 (Case 12, Table I).—K. Y., a man, 28 years of age, was admitted on July 22, 1948, complaining of cough and thick yellow sputum, which was positive for tubercle bacilli. A roentgenogram of the chest, made on admission, revealed a dense shadow including a cavity in right upper lung field, a so-called "lobité supérieure." Right extrapleural pneumonolysis with plastic balls was performed on Nov. 26, 1948. Stripping was difficult due to hard thick pleura and only four balls were inserted. Clinical symptoms remained unchanged after plombage. Because of the positive sputum the removal of the plastic balls and a thoracoplasty was indicated. Direct radiogram showed no changes in the extrapleural space and balls, but preoperative bronchogram revealed lung perforation, which we did not expect (Figs. 1 and 2). On March 23, 1951, the removal of the plastic balls was attempted. As soon as the balls were removed three perforations were found. Because the patient was unconscious during operation, due to excessive use of narcosis (1.0 c.c. of 4 per cent Pantopon-scopolamine and bleeding severely, he aspirated blood through a lung perforation and died from suffocation on the operating table. At autopsy, there were found six perforations of large and small sizes ranging from 1 to 5 mm., which directly communicated to three segmental bronchi of the upper lobe.

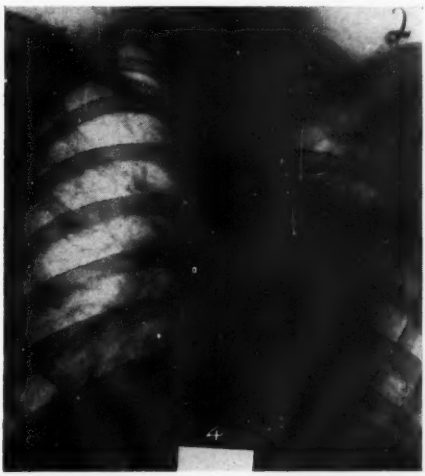
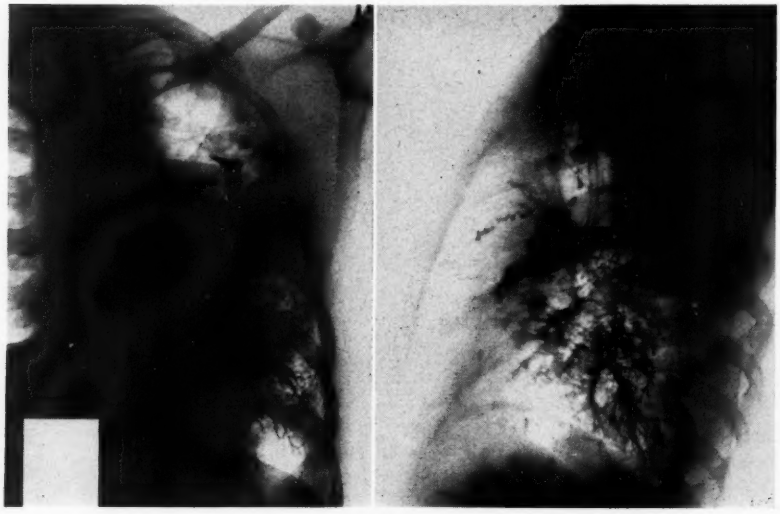


Fig. 3.—(Case 3.) Posteroanterior roentgenogram of chest showing left extrapleural plombage (May 10, 1951).

CASE 3 (Case 22, Table I).—K. M., a 37-year-old man, was admitted on July 9, 1950. He had undergone left extrapleural pneumonolysis with plastic balls in another hospital in July, 1949. Complaints included cough and sputum, positive for tubercle bacilli on direct smear. A roentgenogram of the chest, made on admission, revealed a lung moderately collapsed by Lucite ball plombage and a thin diffuse shadow, suspected of being bronchiogenic spread below the plombage on left side and scattered nodular shadow in right lower lung field. There was no fluid in the balls and extrapleural space, but the membrane between the balls was blurred and thin, suggesting liquefaction. He was occasionally feverish, but the patient's general condition was good. Bronchogram revealed lung perforation (Figs. 3 and 4). On Sept. 21, 1951, removal of the plastic balls was attempted. Operation was easy and six lung perforations were found. Wound was closed primarily. On the ninth postoperative day wound separation occurred and tuberculous mucopurulent exudate was discharged and bronchopleural fistula developed. At present a change of dressing is necessary twice daily. Prognosis is considered poor.

CASE 4 (Case 27, Table I).—F. W., a 25-year-old woman, was admitted on July 28, 1941. Roentgenogram made on admission revealed scattered productive nodular shadow in the left upper lung field. She had a small amount of cough; sputum was positive for tubercle bacilli on concentrated smear. On Sept. 20, 1949, left extrapleural pneumonolysis with plastic balls was performed. After plombage the patient had no clinical symptoms, but the sputum remained occasionally positive. Direct radiograms after plombage showed no change. On Nov. 6, 1951, removal of balls was attempted. Because the membrane between the balls was fragile and partly liquefied, removal was easy. Upon removal of the last ball, situated in the medial part, without any rough handling, massive bleeding abruptly occurred. Since it was considered impossible to control the bleeding, gauze packing and primary tight closure of wound was made. Postoperative course was uneventful except for slight dyspnea until the fourth day. On the fifth postoperative day massive hemoptysis occurred and subsequently daily hemoptysis continued and the patient died of fatal hemoptysis on the twentieth postoperative day. It was thought that a large vessel might have been eroded by plastic balls. Autopsy revealed small perforation of left subclavian artery and a 2 mm. perforation of the lung. Erosion by the pressure of plastic balls on the arterial wall was not detected. The perforation may be attributed to a technical failure in the operation, but if there had not been a lung perforation, she might have survived.



A. *B.*

Fig. 4.—*A*, Posteroanterior bronchogram showing lung perforation (Aug. 8, 1951). *B*, Left lateral bronchogram.

COMMENT

Extrapleural pneumonolysis with plombage is a desirable operation. It is not surprising, therefore, that repeated attempts have been made to find an ideal material for a pack substance. It has been emphasized that plastic is an inert material in the body. However, many of the same late complications appear as usually attend the use of other packing materials. Of these complications, lung perforation is most serious and occurs more frequently with plastic balls than through other packs. Soon after the use of plastic balls for plombage

dense hyaline tissue forms around the balls which is so hard that Trent[2] reported that in one case removal of the spheres post mortem required a mallet and chisel. It is noticeable that this dense hyaline capsule becomes fragile in the course of time and changes to tuberculous granulation tissue in many instances. Sputum conversion after plombage is also less than in thoracoplasty. Our results justify discarding the use of plastic balls in the extrapleural space. Patients who have undergone this operation, even if at present they are at work in good condition, must be periodically observed.

SUMMARY

1. Sixty-three cass of removal of extrapleural plastic balls, in which extrapleural pneumonolysis with filling of plastic balls five months to three years and three months before had been carried out, were analyzed.

2. At operation four lung perforations, two necrotic changes on the pleura, which are considered the beginning stage of perforation, and nine extrapleural empyemas were found.

3. The membrane between the balls had become fragile in the course of time and removal of plastic balls was easy in most cases when more than two years had elapsed after plombage.

4. Rib resection after removal of the balls must be limited to the extent of obliterating the extrapleural space. The resection of five ribs, from the second to the sixth, in one stage is not dangerous. The first rib and transverse process need not be resected.

5. As operative complications, there were three operative deaths, seven sinus formations, and one wound separation. Operative complications occur more frequently than in usual primary thoracoplasty.

6. Preoperative bronchography is useful for detection of bronchopleural fistula.

7. Plastic balls are not suitable for pack material in the extrapleural space.

My hearty acknowledgments are due to Professor S. Tsuda for his guidance.

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PATHOGENESIS OF BRONCHIECTASIS

Experimental Study and Anatomic Findings

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THE problem brought about by the study of the pathogenesis of bronchopneumonectasis is one of the most complicated in pulmonary pathology. Known for many years, this condition has been studied by numerous authors who, on the whole, contributed elements considered of importance for the knowledge of, if not for the pathogenesis of, bronchial dilatations.

Laennec,[1] in 1826, thought that the increase of retained secretions was an important factor in the pathogenesis of such bronchial alterations. Corrigan,[2] in 1838, stated that the sclerosis and condensation of the lung (pulmonary cirrhosis) are alterations which precede the bronchial dilatations. Hasse,[3] in 1841, also agreed with the theory that bronchiectasis appears when the lung decreases in size. Virchow,[4] in 1846, pointed out for the first time that the dilated bronchi are nearly always covered by cylindric ciliated epithelium. Von Biermer,[5] in 1860, compiled in a special paper the available data up to that time on bronchiectasis; he pointed out the mechanical factors (pressure of the retained secretion, increase of the pressure of the air, and pulmonary retraction) and the nutritional factors (pathologic alterations of the alveolar and bronchial walls) which were capable of producing the condition. Stewart,[6] in 1867, pointed out, as Virchow had done previously, the integrity of the epithelium and the destruction of the muscular elastic layers.

Thornton and Pratt,[7] in 1908, diminished the importance of coughing as a factor of the dilatation. In 1923, Nissen[8] produced, experimentally, bronchial dilatations by means of bronchial ligatures in dogs, cats, and guinea pigs; he noted the rapid dilatation which followed the retained secretion of mucus, although infection did not necessarily begin. In 1931, Erb[9] carried out an anatomopathologic study in a large number of cases and stressed the importance of bronchial infection in the origin of bronchiectasis. Robinson,[10] in 1933, after an extensive anatomic study reached the conclusion that the essential alteration was a weakening of the muscular elastic layer, which was produced by inflammatory parietal processes. Jones and Cournand,[11] in 1933, carried out a study of poststenotic bronchiectasis. Warner,[12] in 1935, after a statistical study believed the most important factor to be the bronchial infection. Lemoine,[13] in 1936, assigned great importance to thrombosis and arteriosclerosis of the bronchial arteries. In 1937, Weinberg,[14] carried out tests on rabbits and reached the conclusion that atelectasis and retained secretions are factors of great importance. Jennings[15] (1937) believed atelectasis to be the most important factor and pointed out, for the first time, the existence of reversible bronchiec-

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tasis. In 1937, in an exhaustive study, Andrus[16] analyzed the causes capable of producing bronchial dilatation and denied, categorically, the dilating mechanism of cough. Adams and Escudero,[17] in 1938, carried out an experimental test which had great repercussion. In their tests a partial obstruction was considered the responsible factor in bronchiectasis.

Lander and Davidson,[18] in 1938, stated that the mechanical factors are the important ones and that the infection which destroys the wall must be considered as a secondary event, for bronchograms of patients suffering from bronchiectasis taken during inspiration and expiration show the dilatation and contraction. Hamman,[19] in 1939, believed that the bronchiectasis, which apparently does not follow an obstruction, does not differ from the obstructive type. He stated: "The origin of bronchiectasis can only be satisfactorily explained when bronchial stenosis is demonstrated. In those cases in which the bronchi are permeable, one has to believe that the stenosis has existed, though transitorily."

Fleischner,[20] in 1940, with arguments based exclusively on radiologic findings, gave importance to atelectasis as a dilating factor. Ogilvie,[21] in 1941, in a synthesis including all of the supposed causes of bronchiectasis, mentioned until that time, divided the causes into two groups: defects of the bronchial wall, and various forces which are associated with the bronchial wall. In 1942, Tannenberg and Pinner[22] carried out a number of tests on rabbits and came to the conclusion that atelectasis is not followed by bronchiectasis, unless an infectious process begins. Lisa and Rosenblatt,[23] in 1943, presented anatomic arguments which favored the infection hypothesis. Riggins,[24] in 1943, believed that the cause of the dilatations was to be found in the destructive infection of the wall and that the collapse or retraction of the lobe was a secondary fact. Also in 1943, Vaccarezza and Leston[25] established that the increase of the endothoracic negative pressure is secondary to the bronchoalveolar hypopressure; they did not consider the latter a determining element, or even one that favored bronchiectasis in the obstructive apneumatosis.

Blades and Dugan,[26] in 1944, reported cases of reversible bronchiectasis (which they named pseudobronchiectasis). In 1945, Renault[27] stressed the importance of the alterations of the bronchial wall and came to the conclusion that the principal factor was a profound alteration of the parietal muscular elastic layer. Lander,[28] in 1946, stated that any theory which wished to explain the formation of bronchiectasis must also explain the formation of emphysema; he added that all bronchiectasis is at the beginning reversible, becoming later permanent due to the rigidity of the parenchyma. Mallory,[29] in 1947, in an excellent study, pointed out that any theory explaining the pathogenesis of bronchiectasis must also agree with the explanation of the four constant clinical and anatomic characteristics: (1) why bronchiectasis is very seldom diffuse; (2) why it is not progressive and does not attack other lobes; (3) why it seldom appears as an isolated anatomic abnormality; and (4) why it is an illness of youth and not of old age. Churchill,[30] in 1949, pointed out the importance the mucous secretion may have in bronchial dilatation in cases of stenosis. During the same year, 1949, Carr and his associates[31] stated that the dilatation was only the demonstrable effect of a pathologic process which harms the bronchial and

parenchymatous tissues sufficiently to interfere with the normal function. Fleischner[32] (1949) thought that the atelectasis produced by the obstruction of the small bronchioles was the cause of the bronchial dilatations.

This series of chronologic findings, although not complete, shows how little is known about bronchial dilatations. What is even more apparent is that the destruction of the wall and atelectasis have been considered, in general, the cause of bronchiectasis and not the effect.

It is very difficult to study the pathogenic problem, although we have passed the period when the study of bronchiectasis was carried out as a post-mortem investigation (and therefore corresponded to individuals with severe bronchial infections). At the present time the anatomic study of human bronchiectasis is not useful in obtaining data referring to the primary alterations for the study is performed on lungs which have been removed by surgery and these correspond to patients who present an evident symptomatology and, therefore, established lesions. Added to this is the fact that experimentally only obstructive bronchiectasis may be obtained; it seems that the complexity of the problem would make it practically unsolvable.

Because of the great morphologic similarity between bronchiectasis of the obstructive type and that apparently of a nonobstructive nature, we believe that both respond to the same mechanism. With this in mind, we have carried out experiments in dogs and have also studied twenty cases of human bronchiectasis in surgically removed lobes. With the dogs we hoped to determine: (1) which was the factor responsible for the bronchial dilatation in cases of stenosis; (2) the course of this dilatation; and (3) if the dilatation was permanent or reversible. With the specimens from the human beings we hoped to discover if any elements existed which could confirm or deny the action of a dilating factor coming from the interior of the bronchus.

MATERIAL AND METHOD

Work was carried out on seventy mongrel dogs, weighing approximately 10 kilograms. Intraperitoneal chloral morphine was used as a general anesthetic; the animals were operated upon while intubated and connected to hyperpressure systems. After incising the skin and muscular layers at the level of the fifth left intercostal space, the ribs were separated and the left bronchus was ligated by means of two thick cotton ligatures, only a few millimeters apart. It is of interest to note that in the first dogs operated upon, the bronchus was ligated with only one ligature and the obstruction was only partial; a small orifice, sometimes the size of a pinhead, remained and atelectasis could not be obtained. The wall was closed in layers; the dogs were given penicillin postoperatively. The dogs were sacrificed at different intervals, ranging from a few hours postoperatively to six months postoperatively.

Several dogs were reoperated upon after a time. After removing one of the lobes, the stenosed segment of the main-stem branch, where the ligature had been placed, was also excised and the lumen was re-established either by means of a plastic tube or an evaginating continuous suture. Before doing this, as much mucus as possible was aspirated; after the suturing, the remaining lobe was re-expanded by hyperpressure through the tracheal tube.

On several dogs, before sacrifice, we tried to measure the bronchial pressure below the stenosis by means of a Hamilton manometer but difficulties, attributed to the great viscosity of the mucus, made it impossible to obtain exact readings.

The twenty specimens demonstrating human bronchiectasis were taken from twenty patients whose only illness was the bronchial alteration. Their ages ranged from 18 to 40 years. These twenty lobes were taken from a number of patients without any previous selection, in order that they should possess the characteristics of representative samples. The study was also carried out in some cases of secondary bronchiectasis due to bronchial carcinomas or adenomas.

RESULTS

Bronchial Obstruction in Dogs.—

In some of the dogs it was not possible to produce atelectasis because a total bronchial stenosis was not obtained. Other dogs showed suppurating bronchopneumonic processes, but in the great majority, 52 animals, bronchial dilatations were obtained which presented specific characteristics. These are the only ones to which we will refer.

Six days after the ligature had been performed, the bronchi appeared, macroscopically, dilated and full of an adherent mucus. Due to the great pressure exerted by the mucus on the bronchial wall, a constant factor was the spontaneous and rapid flow of the mucus when the bronchi communicated with the exterior, either by means of the sections of the lung or by means of the opening of the air duct (Fig. 1). In every case the surrounding parenchyma showed atelectasis. The longer the interval allowed before sacrificing the dogs, the greater was the bronchial dilatation obtained; after the second month the caliber of the dilatation became uniform, that is, after the period of two months, the dilatation did not increase and the size remained unchanged in every case.

Macroscopically, no bronchial dilatation was observed before the sixth day following the stenosing ligature, although by means of microscopic observation the presence of bronchial collapse was observed in the first postoperative hours; the dilatations commenced in the following days. The histologic study carried out fifteen hours after the bronchial ligature had been performed, showed the disappearance of the bronchial lumen due to collapse of the walls (Fig. 2, *A*). Once this first condition of bronchial collapse which lasted twenty-four hours, had passed, the bronchi started to dilate due to the continuous secretion of mucus and the interruption of the normal flow to the exterior. This dilatation was obtained during the first postoperative days due to the unfolding of the bronchus. It is necessary to remember that the bronchus of the dog has characteristics similar to that of a human being, but that the mucous membrane in dogs presents numerous folds, which makes the dilatation possible only with the stretching of the epithelium and the flattening of the submucosa (Fig. 2, *B* and *C*).

Some structural modifications became apparent in the epithelium: the number of the goblet cells increased and surpassed the number of the ciliated cells. The epithelium became higher than normal. The bronchial glands showed no alterations and there were no changes in the muscular elastic layers (Fig. 2, *D*). The bronchial lumen was occupied by mucus with some

macrophages and very few polynuclear leukocytes. In the noncartilaginous bronchi, the epithelium was also of a greater height than normal and showed a great quantity of goblet cells. It is of interest to point out that although the bronchial diameter increased up to twice its original caliber, no regressive al-

Fig. 1.—*A*, Transverse section of a pulmonary lobe of dog, three months after a stenosing ligature was produced. Note the protrusion made by the mucus. ×3. *B*, Sections of a lobe of a dog, one month after the stenosing ligature. *C*, Section of a dog's pulmonary lobe twenty days after the stenosing ligature had been produced. The mucus has been extracted to show the bronchial dilatation.

terations of the cells were found and no detachment of the cilia; this indicated a certain capacity of dilatation, for if this were not the case, these elements would suffer alterations due to the pressure to which they were subjected.

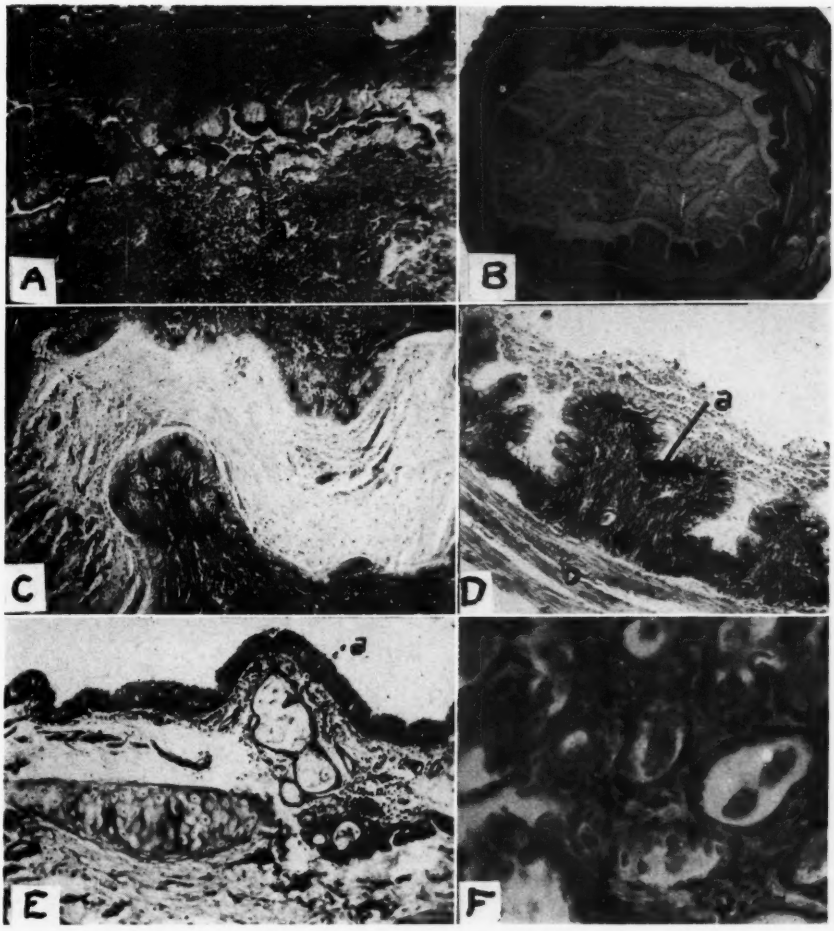

Fig. 2.—*A*, Longitudinal section of a dog's bronchus (*a*), fifteen hours after the stenosing ligature had been produced. A total collapse exists. The surrounding parenchyma is in atelectasis (hematoxylin and eosin, ×150). *B*, Transverse section of a dog's bronchus ten days after the obstruction was produced. Unfolding is nearly complete; structures are conserved, mucus within ×100. *C*, Longitudinal section of a dog's bronchus twenty-five days after the ligature was performed (compare with *A*); total unfolding of the mucous membrane; mucus in the interior (hematoxylin and eosin, ×150). *D*, Specimen of a bronchial wall of a dog twenty days after the bronchial obstruction was produced; *a* shows total compression of the elastic tissue, *b*, normal muscular tissue (hematoxylin and eosin, ×200). *E*, Specimen of a bronchial wall of a dog two months after the obstruction was produced. Total unfolding of the mucous membrane, completely dilated bronchial gland (*a*), disappearance of the excretory duct, its epithelium corresponds to the epithelium which covers the glandular dilatation (hematoxylin and eosin, ×200). *F*, Dilated glands with flattened cells, which correspond to a post mortem carried out on a dog three months after the bronchial obstruction was produced; compare with Fig. 5, *B*.

After a time and once the endobronchial pressure had reached a point in which it inhibited the secretion of the bronchial glands, the excreting ducts of the glands became dilated, and the period of maximum dilatation with glandular exhaustion was attained (Figs. 2, *E* and *F* and 3, *B*). Mucus was found not only in the bronchi and bronchioles, but also in the alveolar ducts; it was an interesting finding to see that a connective tissue organization of the mucus was started in the bronchioles (Fig. 3, *A* and *C*). After about two months had elapsed since the stenosis was produced, the submucosa became infiltrated with round cells, lymphocytes in their greater part, and within a very short time a true cuff surrounded the air ducts.

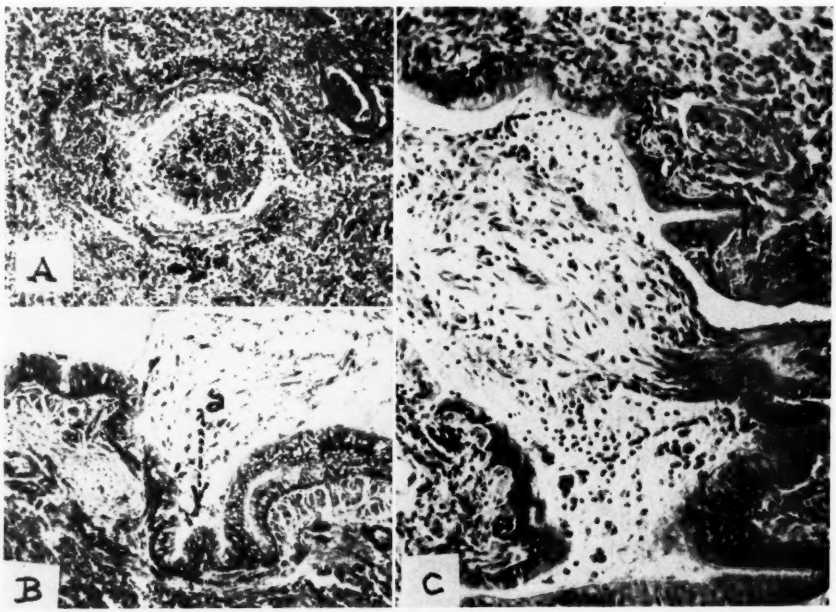

Fig. 3.—*A* and *C*, Bronchiolar organization of the mucus. In *A*, within a small bronchiole, a nodule infiltrated with lymphocytes may be seen (dog three months after the obstruction was produced) (hematoxylin and eosin, ×150). *C*, Small bronchus of a dog two and one-half months after the obstruction. Mucous organization. Note the fibroblastoid cells which seem to come from the bronchial epithelium (hematoxylin and esoin, ×250). *B*, Bronchus of a dog, one month after the obstruction. Total unfolding with apparent disappearance of the submucosa. *C*, Corresponds to an amply dilated excretory duct.

In summarizing the observations made from the dogs, it may be stated that once the bronchial stenosis was established, the air was reabsorbed from the respiratory parenchyma and also from the bronchi. As the mucus, which continued to be secreted by the bronchial glands and the goblet cells, did not find an external outlet, it dilated the bronchus. This was made possible, first by compression of the mucous membrane and flattening of the chorion. Later there was dilatation of the excreting ducts, until a time was reached (approximately two months) when the pressure of the mucus within the bronchus was higher than the resistance offered by the cells of the acinus which ceased to secrete mucus. Thus the dilatation of the bronchi and bronchioles became

stabilized; then the connective tissue organization with bronchiolar obstruction commenced in the small bronchioles and in some small bronchi. We will refer later to the importance of this latter finding.

In those instances where the lobe was removed, changes similar to those just listed were observed. Survivals after recanalization were obtained and were of two, six, nine, and ten days. Difficulties of a technical nature made a correct recanalization impossible. The remaining lobe showed atelectasis in two dogs, in another an inflammatory process was added and the dilatations remained as a definite bronchiectasis (Fig. 4, *A*). In the dog that survived ten days, a partial recanalization was obtained, although the bronchial dilatations persisted as such in the absence of any infection (Fig. 4, *B*).

Fig. 4.—*A*, Sections of a dog's lung several days after the recanalization of the bronchus, which had been obstructed five months. Definite bronchiectasis may be observed, as an inflammatory process set in. The lobe, removed for control purposes, showed at the time of the recanalization, mucous bronchodilatation. *B*, Dog's lung ten days after the bronchial recanalization was attempted. The obstruction existed during six months. The bronchial dilatations did not show any accompanying inflammatory processes. The lobe extracted for control purposes, showed mucous bronchodilatation, ×3.

Even though this last group of animals was small and the results obtained, due to technical difficulties, not too clear, the results do show that infection superimposed to the dilatation produced by retained secretions, led to the production of bronchiectasis and that in some cases, even though the dilating material was extracted, the bronchus remained dilated.

Human Bronchiectasis.—

Of the twenty patients studied only four showed serious inflammatory processes. In the remaining sixteen slight or no inflammatory processes were observed. In the majority, parenchymatous alterations existed which consisted of sclerosis, atelectasis, emphysema, and bronchial proliferation; in very few the parenchyma presented normal characteristics.

The microscopic examination of the bronchiectasis showed:

Epithelium.—Practically all the bronchi presented epithelium of the cylindric ciliated pseudostratified type, with a great quantity of goblet cells and was,

in general, of a greater thickness than the normal lining epithelium. Even in the small and dilated bronchi, the epithelium showed hypertrophic characteristics. This is a histologic fact that repeats itself with extraordinary constancy in all bronchiectasis without infection.

Bronchial Submucosa.—In the large bronchi, the submucosa was thinned and showed some zones where the epithelium laid directly on the cartilage. In the small bronchi a lymphocyte infiltration of a varied degree was observed in the submucosa and peribronchus.

Elastic and Muscular Tissues.—In general, in those cases without infection the characteristics were normal. We omit the description of small details of thickening, fragmentation, and reduplication of the elastic fibers which some authors have observed and which we corroborate, even though they lack importance.

Cartilage.—In all dilated bronchi, cartilaginous alterations of the regressive type were found: calcification, ossification, and sometimes even formation of bone marrow.

Peribronchus.—The condition of the peribronchus varied; in some cases sclerosis was observed and in others the characteristics were normal.

Bronchial Glands and Excretory Ducts.—In these elements histologic findings of the greatest importance were observed. The glands, according to the degree of dilatation attained by the bronchus, were normal or completely atrophied, passing through degrees of true acinous dilatation with formation of small cysts. In the bronchi with a greater dilatation it was difficult to individualize the glands, while in those cases in which the dilatations were not great the glands showed characteristics similar to normal; other intermediate cases existed in which the acini were full of mucus and the cells flattened (Fig. 5, *B*). The corresponding excretory ducts showed alterations related to the glandular alterations, from normal to completely dilated; there existed some zones, easily individualized, in which, as a result of the great dilatation, the epithelium of the duct formed part of the bronchial epithelium. As a result of these dilatations of the excretory ducts, either partial or complete, the underlying bronchial submucosa and epithelium took on the appearance of papillae (Fig. 6, *A* and *B*).

The bronchial arteries showed, in general, a thickened wall and hyperplasia of the intima, of the disused arteritis type. This fact has been observed by the majority of those studying bronchiectasis, although receiving different interpretations.

To summarize, we may say that in cases of bronchiectasis without a serious infection, which constitute the great majority of cases, the histologic examination generally showed normal epithelium and musculoelastic structures, lymphocyte infiltration of the submucosa, and alterations (of a possible anoxic nature) of the bronchial cartilage. Sometimes there were rigidity of the peribronchus and dilatation of the glandular excretory ducts.

In the pulmonary parenchyma which surrounds the bronchiectasis, obstructive pneumonitis was found which alternated with areas of emphysema. The bronchioles were dilated and many showed mucous bronchodilatation.

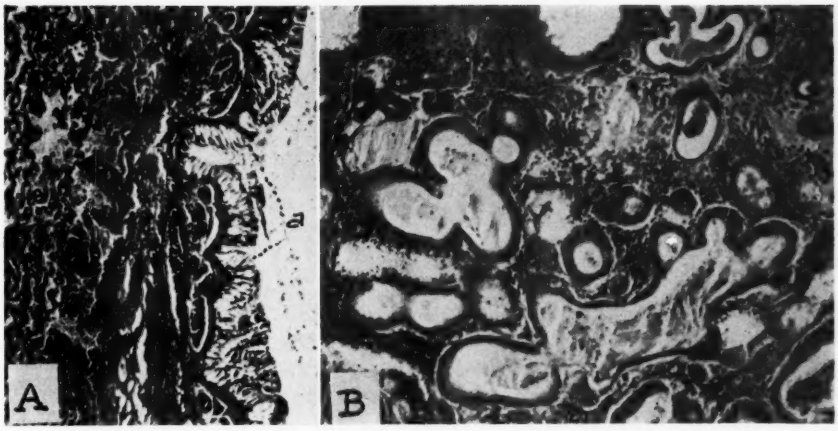

Fig. 5.—*A*, Wall of a human bronchiectasis, total unfolding of the mucosa (large quantity of goblet cells (*a*), apparent disappearance of the submucosa (hematoxylin and eosin, ×180). *B*, Human bronchiectasis. Dilated glands which contain mucus and flattened cells (hematoxylin and eosin, ×200). Compare with Fig. 2, *F*.

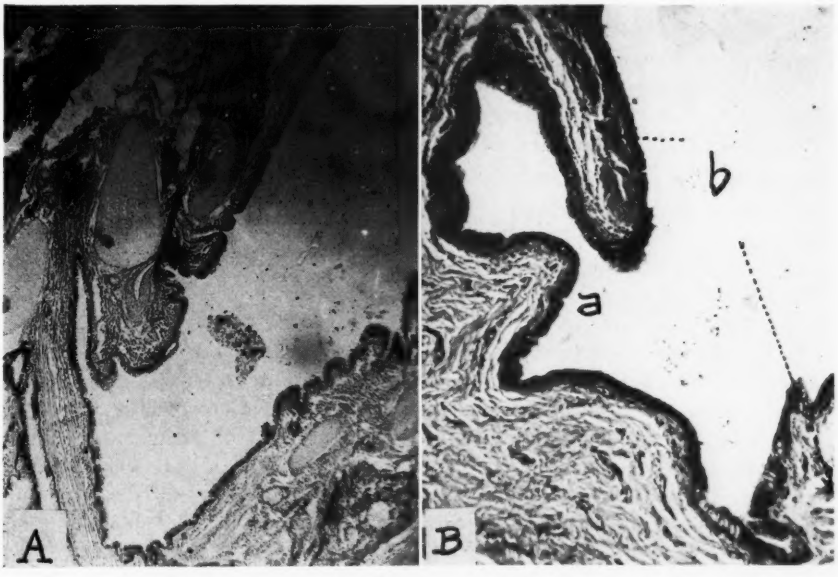

Fig. 6.—Human bronchiectasis. Wall without an inflammatory process, dilatation of excretory ducts. Notable thinness of the submucosa (hematoxylin and eosin, ×30). *B*, Segment of a human bronchiectasis' wall. Normal epithelium; apparent disappearance of the submucosa. Markedly dilated excretory duct (*a*). Absence of muscle in the zone of the duct and its presence at both ends (*b*), formation of a papilla (hematoxylin and eosin, ×200).

DISCUSSION

The results obtained from the experiments with dogs and from the study of the material of human bronchiectasis allow us to answer the questions we asked at the beginning of this paper.

The Responsible Factor of Bronchial Dilatation in Cases of Stenosis.—Even though the atelectasis was supposed by many authors to be the cause of the dilatation, it is only a contemporary factor of the process, but without any causal importance. In fact, once a bronchial obstruction is obtained, the atelectasis is produced by diffusion of the alveolar air and reabsorption of the air which remains enclosed in the bronchi below the stenosis. As the secretion of mucus by the bronchial glands is not interfered with, and since this secretion does not find an outlet to the exterior, the bronchus becomes dilated. At the beginning the arrangement of the structures of the mucous membrane makes the dilatation possible without any alteration of the underlying structures. In the short time of six days, dilatations of macroscopic appearance are obtained. For descriptive purposes we will call these dilatations "mucous bronchodilatations" and list them as the first condition of all obstructive bronchiectasis. We refer, as an example of this statement, to a case of carcinoma located in the bronchus of the left lower lobe, in which the intrabronchial growth obstructed first the bronchi of the posterior and medial basal segments where there existed, at the time when the study was performed, definite bronchiectasis. In its later growth the tumor obstructed the bronchus of the anterior segment, and in this segment mucous bronchodilatations were observed surrounded by atelectatic parenchyma, similar in every way to the observations in dogs (Fig. 7, *A* and *B*). Also as an example, we offer the cases of bronchial adenoma in which below the stenosis great mucous bronchodilatations are found and when an infection is added, bronchiectasis is produced. (Fig. 7, *D.*)

Evolution of the Dilatation Beyond the Stenosis.—In dogs, the dilatation progresses until maximum dilatation is reached (after approximately two months), in which the glands are inhibited and the flow of mucus ceases. While the large bronchi dilate there is a flow of mucus from the glands; but once the bronchi cannot dilate any further, the flow ceases and organization of the retained mucus begins (Fig. 3, *A* and *C*). We do not know what mechanism leads to this proliferation of mesodermic and mesenchymatic elements, the final result of which is the total or partial bronchiolar obstruction, but it may be likened to what occurs in the blood vessels in cases of detention of the blood flow and also with the organization which follows the deposit of fibrin on the serous surfaces. The consequence of this bronchiolar obstruction will be the inability to re-expand when the obstruction becomes complete and generalized, and emphysema as well as incomplete re-expansion when it is neither complete nor generalized.

Is the Mucous Bronchodilatation Reversible or Permanent?—Jennings,[15] Blades and Dugan,[26] and Ochsner[33] reported cases of reversible bronchiectasis. We do not doubt that such a thing may occur since nothing prevents mucous

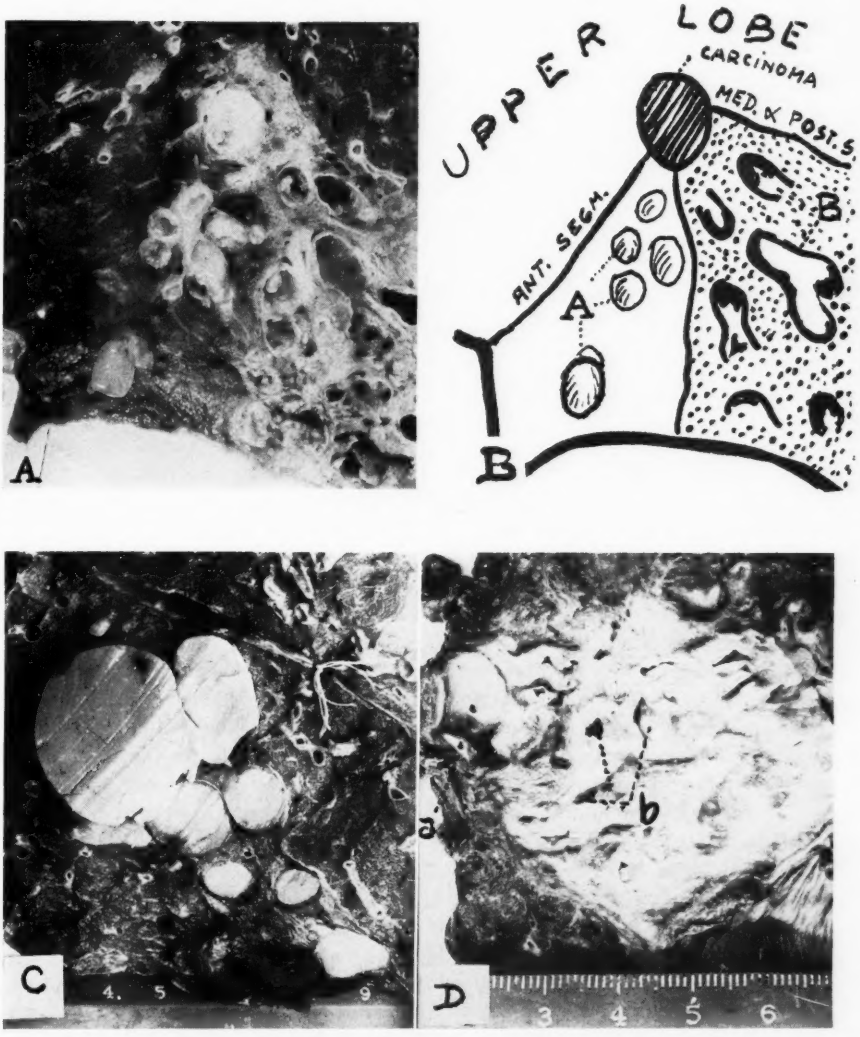

Fig. 7.—*A* and *B*, Carcinoma of a bronchus of the left inferior lobe, definitive bronchiectasis in the basal, medial and posterior segments (*B*), mucous bronchodilatations in the anterior segment (*A*). *C*, Mucous bronchodilatation in a case of bronchial obstruction caused by an adenoma. *D*, Bronchial adenoma (*a*) and definitive bronchiectasis (*b*) by an inflammatory process.

dilatations from being a reversible process in the early stages, as at the beginning the bronchus can become dilated and there are no bronchial or parenchymatous lesions. After some time, two months in the case of dogs, it is difficult for these dilatations to be reversible for bronchiolar fibrous obstructions and destructive alterations of the bronchial glands are added. As a consequence of this, the total re-expansion of the lobe does not take place, and emphysema and obstructive

pneumonitis occur when the stenosis of the bronchioles is not total. Due to the glandular destruction the infection of the bronchial wall is made possible.

Technical difficulties did not allow us to obtain conclusive results with regard to the reversibility of the mucus bronchodilatations. A dog, on which a lobectomy was performed six months after and in which a partial pulmonary recanalization was obtained, maintained its bronchial dilatations (Fig. 4, *B*); another, subjected to the same experiment, survived only a few days after the lobectomy and, as a consequence of the added inflammatory process, the bronchodilatations became definite bronchiectasis (Fig. 4, *A*). In the other dogs a good reexpansion was not obtained, and although at the time of the lobectomy the greatest possible amount of mucus was extracted, the bronchi persisted dilated for several days.

We believe, however, that the mucus bronchodilatations are reversible processes at the beginning, irreversible after a certain time when parenchymatous alterations and inflammatory processes of the walls are added.

Findings Which Affirm, in Human Bronchiectasis, the Existence of a Dilating Factor Proceeding From the Interior of the Bronchus.—The alterations encountered in human bronchiectasis lead us to believe that the inflammatory lesions of the bronchial walls are secondary and are of no importance or value at the beginning of the bronchial dilatations. This is based on the observation of sixteen patients of a total of twenty, who did not present inflammatory lesions; this is also in accordance with observations made by other authors.

All alterations found in the bronchi tend to show that the increase of the internal pressure is the cause of the bronchial dilatation. To use an example which would give a clear idea of our observations in bronchiectasis, we can say that it would be as though the normal bronchus were strongly inflated from one of the ends, and as a consequence the folds would be ironed out. The findings cannot be explained by external stretching but only by the increase of pressure from within.

Therefore, our findings would indicate the possibility that apparently nonobstructive bronchiectasis is the consequence of a previous obstruction. The reason that in some cases of definite bronchiectasis the obstructing element is not found, is not, we believe, an important argument to rule out the hypothesis for, as has been observed in dogs, the rapidity with which the mucous bronchodilatations begin is remarkable. If we bear in mind that the bronchiectasis represents processes localized to one or two lobes and which do not extend to other lobes, and that in certain cases they are reversible, it is well understood that there may exist a transitory obstructive element which disappears, leaving reversible or definite bronchiectasis.

We have expressly left for the end, one point of discussion which we believe upholds the obstructive mechanism of the primary human bronchiectasis. We refer to bronchial proliferation. This name is given to small ducts of an irregular contour, covered by cuboid epithelium, placed in the center of the pulmonary parenchyma which has undergone a process of postatelectatic sclerosis. Its significance is difficult to understand. Some authors give it the value of a collateral aerial circulation.[34] This must not be mistaken for the

lining of the alveolus. It is of a completely different origin. In the case of the fifty-two dogs used for the study, we never found it, while we came across it in the twenty examples of human bronchiectasis studied. This finding which seems a discordant fact, actually, is not. For the animals presented definite atelectasis, and when in some of them the bronchial obstruction was relieved the later survival was short; a useful re-expansion was not obtained. In the twenty human beings studied, however, there did not exist any bronchial obstruction at the time the study was made. That is, that if both groups are in a certain manner comparable, the one referring to human bronchiectasis is a much later condition. If we consider that apart from this bronchial proliferation, great areas of sclerosis and disappearance of bronchioles are found, as was mentioned by Rokitansky[35] more than a century ago and recently by Reid,[36] we are led to the belief that the bronchiolar obstruction is the cause of the segmentary atelectasis and the bronchial proliferation is a phenomenon which follows re-expansion. We, therefore, believe that bronchial proliferation, constant in human bronchiectasis, indicates the previous existence of atelectasis and a later recanalization. This bronchial proliferation occurs not only in bronchiectasis, but is observed in all cases of partial atelectasis. It is not observed when a permanent atelectasis exists, such as in the cases of the dogs studied.

The problem to be solved at this time is to find out which is or which are, the cause or causes capable of obstructing the bronchi without leaving any traces.

CONCLUSIONS

1. Complete bronchial obstruction leads to bronchoalveolar apneumatosis. The secreted mucus, not finding an external outlet, dilates the bronchus (mucus bronchodilatation).

2. The mucus bronchodilatation constitutes the first phase of every obstructive bronchiectasis.

3. The mucus bronchodilatations may, once the bronchus becomes recanalized, disappear at the beginning, or become permanent later (bronchiectasis) as a result of the bronchiolar obstruction or as a consequence of an accompanying infection.

4. It is probable that all bronchiectasis, acquired, primary or secondary, is caused by a bronchial obstruction.

We are indebted to Dr. Oscar Vaccarezza, Chief Thoracic Surgeon, for his valuable suggestions and the material dealing with bronchiectasis in human beings; we also acknowledge the technical assistance of Mrs. M. L. O. de Caraglio and Miss Guisella Feverman.

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RATIONALE AND A NEW SURGICAL TECHNIQUE IN TRAUMATIC CHYLOTHORAX

With Report of a Case

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WE PRESENT a re-evaluation of the treatment of traumatic chylothorax and a few ideas concerning the definitive surgical technique that may be utilized for injuries to the thoracic lymphatic system.

Portrayed is the situation with which one may be faced and how simple mechanically the solution may be.

The following tenets are proposed:

1. Assiduous clinical and roentgenographic follow-up of cervical, thoracic, and abdominal injuries, whether penetrating in type or not, is necessary.

2. There should be close nutritional support to compensate for loss of chyle.

3. If daily completely evacuant thoracenteses do not alleviate cardiac or respiratory embarrassment, or leakage of chyle does not subside, we believe in closed (tube) drainage with suction applied. Despite the alleged bacteriostatic property of chyle, a thoracostomy tube or tubes should not be present longer than seven to ten days lest empyema supervene.

4. If the lung does not expand fully, thoracotomy should be considered seriously. A "golden period" of seven to fourteen days after trauma exists for operative attack before inanition and collapse occur. Suture of a leak in a bronchus or the pulmonary parenchyma, a plastic procedure on the duct, or simple ligation may turn the tide. The normal apposition of visceral to parietal (mediastinal) pleura may be all that is necessary, and the cohesive, adhesive, and tamponade effect of the fully inflated lung may be curative in itself.

INCIDENCE

Up to 1895, Bargeduhr[49] had been able to collect only forty cases, and he estimated that one occurred every six years. Felts[18] declared that many clinicians of wide experience have never encountered a case. Military surgeons reported no such case during the Civil War, according to Lillie and Fox.[24] We found no mention of chylothorax in *The History of the Medical Department of the United States Army in the World War.* Berry[5] has discussed the only case reported as occurring in the American Army during World War II. Until 1935 only eighty cases were reported.[24] Olsen and Wilson[33] revealed that until 1944 only nine such cases were treated at the Mayo Clinic. While reviewing the literature, Whiteside[48] found that fifty-four cases were in the journals prior to

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1918 and forty-five during the period from 1918 to 1949. During the 1949 convention of the American Association for Thoracic Surgery, six additional cases were discussed, according to Birt.[7]

HISTORY

Macnab[29] reported that Asellius in 1627 was the first to describe the lymphatic system. Thirty-six years later, Longelot (Cellan-Jones[10]) wrote of the clinical entity of chylothorax. The first authentic case of traumatic chylothorax was detailed by Qincke in 1875 (Lillie and Fox[24]). Intravenous chyle in the therapy of this discouraging catastrophe was first administered by Oeken in 1908 (Birt[7]). We believe the first case in the English literature to be that of Williamson in 1920.[49] Van Nuys[46] in 1931 reported the use of roentgen therapy. First to report bilateral chylothorax with associated chylous ascites of traumatic origin was Brown[8] in 1937. During the same year, Bauersfeld[3] published a report concerning the second patient to receive chyle by the intravenous route and the first so treated to survive. Peet[34] in 1943 was the first to inject chyle into the sternum. It was as recent as 1946 that Lampson (Meade and associates[31]) proved that the condition could be cured by ligation of the duct in the chest. From 1946 to 1951, five cases of transthoracic duct ligation were reported by Shumacker.[37]

SCIENTIFIC BACKGROUND

Anatomy.—Anatomists mention practically nothing concerning the cross-sectional dimensions of the duct or its lumen. Cunningham[13] stated that the total length of the duct averages about 45 cm. It is dilated at both its origin and termination. The narrowest point is usually opposite the fifth thoracic vertebra. The caliber is variable, and often the thoracic part consists of a series of anastomosing channels. The widest part is the cisterna, which may be entirely absent. Several valves are present and formed by semilunar folds of the tunica intima, arranged in pairs, and the most perfect pair is situated at or near the lymphovenous junction. Agreeing with the anatomists, Thorek[44] stated "At the level of the seventh thoracic vertebra the duct begins to cross obliquely to the left until it reaches the left side of the body about at the level of the fifth thoracic vertebra."

Histology.—The duct has been described by Bailey[39] as possessing a considerable amount of muscle tissue. "The intima consists of an endothelial lining, a thin intermediate layer of fibro-elastic tissue in which are bundles of longitudinal muscle fibers, and an internal elastic membrane composed of a longitudinal network of elastic fibers. . . . The media is the thickest coat and consists of longitudinal and circular muscle bundles, the former predominating. . . . The adventitia is poorly defined. Near the media is a layer of coarse collagenous fibers, mainly longitudinally disposed and containing considerable elastic tissue and occasional longitudinal muscle fibers. The outer layer is more finely fibrillar and merges with the surrounding areolar connective tissue."

Physiology.—In discussing the paper by Shackelford and Fisher, Penick[19] stated that valves in the duct are inefficient and that extraductal pressure could force chyle in both directions. Cummins[12] made the interesting comment that in his case the duct was collapsed to the size of a No. 30 linen thread and that after ligation the ductal diameter was that of a No. 6 catheter (English). This leads us to assume that the application of pressure against the duct caudad from a point of leakage and against a centrum may make this duct easier to delineate with or without opening the mediastinal pleura. It has been stated by Drinker and Yaffey[15] that after ligation the intraductal pressure may reach 35 mm. H_2O.

The regulatory factors affecting the flow of chyle as given by Birt[7] are as follows:

1. The vis a tergo effect depends upon the volume arising from the intestinal lymphatics.
2. There is a pressure gradient between the chest and abdomen. Inspirational negative pressure draws chyle into the chest, and the descending diaphragm increases the pressure on the abdominal lymphatics.
3. Vagal stimulation contracts the smooth muscle in the duct with a decrease in flow. Main[30] concurred.
4. A vacuum pump effect exists in the jugular vein (we assume that Birt had in mind the side-arm or Venturi principle of suction). In treatment Birt believed it logical, therefore, to induce oral starvation, administer intravenous fluids, use gastric suction to immobilize the intestines, and perhaps administer acetylcholine.

We are somewhat confused by the statement made as recently as 1952 by Berthrong and Pike[6] that "lacerations of the thoracic duct clinically and experimentally do not result in continued chylous drainage or chylothorax—the thoracic duct has normally many collateral channels so that thrombotic occlusion of damaged vessels usually does not result in a rise of lymphatic pressure and leakage."

Bacteriology.—Birt[7] said that chyle appears to be bacteriostatic. This was suggested by Lampson in 1948. We have read of no one being able to culture any organism from chylous fluid in this clinical entity, and we were not able to do so in our case.

Pharmacology.—Birst also stated that vagal stimulation produces contraction of smooth muscle and decrease in flow of chyle through the duct (and also through a point of leakage) and that Adrenalin produces dilatation of the duct and an increase in flow.

ETIOLOGY

This variety of chylothorax has been caused by a variety of injuries, accidental and surgical. Cases have been reported as due to the following causes: knife wound of the neck[25, 47]; compression of the trunk[1]; contusions of the back[24, 29]; auto accidents[3, 6, 8, 22, 50]; fracture of the left clavicle and injury to the left subclavian vein[4]; contusions of the chest[7, 20]; fracture of the ribs[14];

gunshot wound of the chest[5, 36, 48]; surgical traumas in general[16]; after splanchnicectomy[23, 34]; after surgery for tetralogy of Fallot[37, 48]; after thoracoplasty with apicolysis[12]; following traumatic pseudocyst of the pancreas[45]; after cough[11, 26]; after hyperextension of the spine[32]; after heavy lifting[9]; and after contusion of the back with fracture of centrums.[10] There may be associated disease causing fixation of the duct to the vertebrae and producing vulnerability. Meade[32] believed fragility of the duct to be an important component.

MORTALITY

In 1938, Shackelford discussed forty patients, nineteen of whom died. Florer and Ochsner[19] in 1945 wrote, "Prognosis is grave since there is a 50 per cent mortality in cases in which operation has not been done, and direct surgery to repair the duct has been completely unsuccessful, resulting in 100 per cent operative mortality." Schaffner and Kirkpatrick[35] stated in 1951, "Such surgical treatment (ligation) has resulted in a marked decrease in mortality and nullifies Cushing's statement 'It can't be done.'" Deaths have been due primarily to respiratory or cardiac embarrassment and severe malnutrition.

MORBIDITY

Tremendous amounts of nutritive fluid are lost, and thoracenteses may be necessary over a protracted period of time. The patient treated by Bauersfeld[3] lost 22,275 c.c. of chyle by aspiration in sixteen days. The patient of Gordon[20] was admitted to the hospital in August, 1937; by December, 1938, aspiration was required once each month. Felts[18] found it essential to tap his patient's chest every three months, twenty months after treatment was initiated. Collapse can occur suddenly, according to Wright,[50] and he advocated frequent examinations to exclude pleural effusion after crushing injuries of the chest and thoracic spine in order that such examinations may result in diagnosis before the onset of pressure symptoms.

CLINICAL COURSE

Fluid from the chest initially may be bloody, in the experience of Lillie.[24] Typically the specific gravity is over 1.012; the fluid does not coagulate; present are microscopic fat globules soluble in ether and receptive to sudan III stain; the fat content is high; and rapid reaccumulation occurs. Lillie stated further that unless fluid is removed, cardiac and respiratory embarrassment occur. If the wound in the duct fails to close, or if collateral fails to become established, malnutrition and emaciation supervene.

To the knowledge of Florer and Ochsner,[19] a relatively asymptomatic interval exists between the trauma and the onset of marked dyspnea, pallor, cyanosis, thready pulse, and shock, and this period averages four days. The time lag in the famous case of Beatty, which was due to fracture of the left clavicle and laceration of the subclavian vein, was six and one-half years. Macnab,[29] one of the oft-quoted authorities, found it difficult to explain the latent period.

Lowman[15] wrote of a diagnostic triad: roentgenographic indications of a rapidly accumulating pleural effusion; low serum protein; and a falling lymphocyte count.

TREATMENT

This may be best discussed by tabulating years, writers, methods, and comments to indicate the trend:

1921 Guldmann[21]: Rectal feedings and thoracenteses.

1926 Stewart and Linner[41]: Thoracenteses; made unsuccessful attempt to dissect the thoracic duct at autopsy.

1929 Andrews[1]: Aspirations of chest with fatal result. Autopsy revealed rupture of duct between eleventh and twelfth thoracic vertebrae. "It is obvious that treatment is unsuccessful. Due to lack of any means for locating the point of rupture and the inaccessibility of the thoracic duct, radical surgery is highly impractical."

1931 Van Nuys[46]: Aspiration. "Every puncture is a damaging factor as regards healing since restored negative pressure caused by aspiration sucks at the wound and makes healing impossible." He empirically gave high-voltage x-ray therapy to the lower mediastinum and believed that such treatment helped the patient.

1934 Heppner[22]: Daily thoracentesis. Autopsy revealed interruption in the continuity of the duct with fibrosis about the ends.

1935 Lillie[24]: Conservative aspiration, intravenous glucose with saline solution. "Ligation in the midthorax is almost certainly lethal. . . . Some favor extensive thoracotomy or thoracoplasty to produce positive pressure in the pleural cavity favoring spontaneous closure of the duct."

1936 Strauss[42]: (a) Desired to have fluid in the chest under pressure for tamponade effect but concluded that without fixation of the mediastinum this could not be achieved; (b) elimination of fat from the diet is important; (c) water intake should be limited; (d) there is no way of producing tamponade by air or fluid; (e) oxygen tent is of no aid when collapse occurs from mediastinal displacement due to collection of fluid in the thoracic cavity; (f) frequent aspiration is necessary to save life; and (g) there is no advantage in leaving some of the fluid. In fact, partial aspiration means more frequent aspiration and every puncture adds to the risk of infection.

1937 Brown[8]: Performed posterior mediastinotomy (tenth thoracic to first lumbar vertebrae) with closed (tube) drainage. Patient died next day from adrenal apoplexy.

Bauersfeld[3]: First patient treated with intravenous chyle in which recovery occurred, and the second reported as receiving chyle by this method.

1938 Shackelford[38]: "Believe evidence is sufficient to contra-indicate feeding of fats during leakage thus reducing intraductal pressure and through relative collapse of the vessel encouraging its repair. Surgical ligation of the duct is impractical."

1940 Gordon[20]: Operative repair is not feasible (nor is ligation) because the patient is in poor condition and the site of rupture of the duct is rather inaccessible technically.

1941 Smith[40]: "Avoid open external drainage of chyle unless empyema supervenes. Chyle should be aspirated only to relieve distress for its accumulation under pressure probably helps to close the fistula."

1943 Schnug[36]: Gave intravenous chyle and on the fifteenth day performed a phrenic nerve operation. Substituted heparin for sodium citrate as an anticoagulant in the chyle; also injected sterile broth into the pleural cavity to stimulate fibrous reaction as reported by Rienhoff. Believed diet and phrenicotomy had no effect and that the value of intrapleural broth is unknown.

Peet[34]: Performed ligation of the duct on the fourteenth postsplanchnicectomy day with immediate fatality and with no satisfactory explanation of the death.

1945 Florer and Ochsner[19]: Considered phrenemphraxis and pneumoperitoneum with the intent of having an elevated diaphragm occlude the rent in the duct.

1947 Berry[5]: There was an associated laceration of the left lung. Treatment was daily aspiration other than repair of laceration without surgery of the duct. Believed surgery of the duct to be difficult and should be used only as a last resort.

1949 Whiteside[18]: Performed transthoracic ligation; recommended morrhuate and fibrin foam locally.

1950 Meade[31]: Believed intravenous chyle should not be used until further experimental investigation of causes of reaction has been made. "One-half of cases reported have recovered from aspiration alone and surgery seems unnecessary." Conduct aspiration at frequent intervals, and initiate closed (tube) drainage if not successful. One must re-expand the lung; if not possible and the leak continues, one must explore. Two weeks should be the maximum interval.

1951 Auld and Needham[2]: "When neurtal fat was given with Sudan III, a substantial amount of dye was recovered from the chylothorax. Whereas, when equivalent amounts of fatty acid and glycerol were given (with dye) no dye was recovered in one instance and only a small amount in another." This suggests feeding fatty acids and glycerol rather than a complete fat.

Schumacker[37]: "A continued loss . . . of large quantities of chyle presents a very difficult nutritional problem. For this reason it seems unwise to postpone surgical intervention until the patient's failing nutritional status makes him a poor surgical risk. . . . The duct can be ligated without harm because of the presence of collaterals."

Schaffner and Kirkpatrick[35]: Recommended low surgical ligation of duct through a right thoracotomy regardless of the location of the effusion, right or left, and agreed in general with Meade.

1952 Meade[32]: "Resect a portion of the duct and ligate the ends—study the resected specimen."

Hoffman and associates[23]: "Ligation of the thoracic duct in the lower third is not considered safe because collateral circulation is not as rich as in the upper third and rupture from high intraductal pressure is brought on by acute obstruction."

Effler[16]: Discussed the disadvantages of surgery and described it as a serious undertaking; stated that finding chyle leaks is a real problem; that there are anatomic variations; that there is a deposition of fibrin and clot; and that extensive mediastinal dissection may convert a unilateral to a bilateral chylothorax.

Birt[7]: Utilized intravenous chyle (fresh effusion which was filtered and then heparin added) and right phrenemphraxis. Declared that if effusion persisted undiminished after phrenic nerve crush that ligation should be undertaken. Also mentioned that Klepser gave green coal-tar dye in olive oil by duodenal tube several hours before operation. Believed that anastomosis of the duct to a near-by vein as described by Hodge and Bridges was needlessly hazardous and difficult since simple ligation will cure.

REPORT OF A CASE

A 19-year-old white male soldier was stabbed over the shoulders and chest Nov. 26, 1952. The following day he was admitted to a civilian hospital where the diagnosis was made. Treatment included pressure bandage to sucking wounds of the thorax and thoracenteses, each of

which procured 1,000 to 2,000 c.c. of fluid presumed to be chyle. On Nov. 30, dyspnea and dull right chest pain appeared, chest roentgenogram revealed obliteration of the right lung shadow, and 1,500 c.c. of fluid was removed.

He was admitted to the hospital at The Armored Center, Fort Knox, Ky., on Dec. 9 with obvious acute distress and dyspnea. With each cough, a large bulge appeared over the right sixth intercostal space in the posterior axillary line. Multiple small penetrating wounds were present over the back, chest, and left upper extremity. Treatment included penicillin and streptomycin, fat-free diet, pressure bandage to hernia, supplemental vitamins, and daily thoracenteses. Smear and culture of the aspirated chyle were negative for bacteria. The neutral fat content was 1.68 mg. per cent, free fatty acid 540 mg. per cent, and cholesterol 50 mg. per cent.

Air evacuation to a chest center was contemplated. However, the rapid reaccumulation of fluid and the possible unavailability of a pressurized cabin plane led us to feel we should provide definitive care.

Fig. 1.—Posteroanterior film of chest Dec. 10 (fourteen days postinjury).

It was determined early that retrogression prevailed. On Dec. 15 (eighteenth post-traumatic day), exploration of the right chest was performed. Early in the morning of surgery a Levin tube was placed transnasally into the stomach, and 240 c.c. of half-and-half mixture of milk and cream instilled—the tube was retained to permit use of dye if found essential. The herniated parietal pleura was incised and twenty minutes was devoted to decompressing the pleural cavity of chyle slowly. The intercostal hernial defect was 8 cm. long. Entry was made by resecting a large segment of the seventh rib. The lung was 50 per cent collapsed, obviously due to a 5 cm. wound in the costal aspect, lateral segment of the middle lobe. This was promptly closed and the lung retracted anteriorly. Milky fluid was seen to exude from a 1 mm. opening in the mediastinal pleura in the general area of the thoracic duct and opposite the posterior extremity of the sixth intercostal space. The esophagus, aorta, azygos, cavae, pulmonary vessels, tracheobronchial tree, and sympathetic chain were undamaged and were protected throughout the procedure. A mediastinal pleural disc, 1 cm. in diameter, with the fistulous opening as its center was dissected free sufficiently and

circumferentially to allow closure of such an inverted cone with a figure-of-eight suture of 0000 silk. The loss of chyle was immediately and completely controlled in this manner without ligation of the duct proper. It was anticipated that the tamponade effect of the expanded and repaired lung would ensure complete success. However, a strip of Gelfoam was

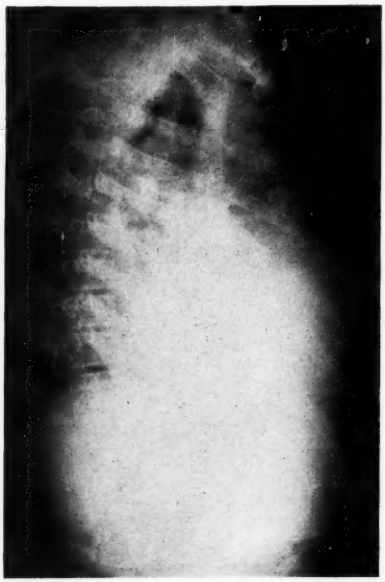

Fig. 2.—Right lateral film of chest Dec. 10.

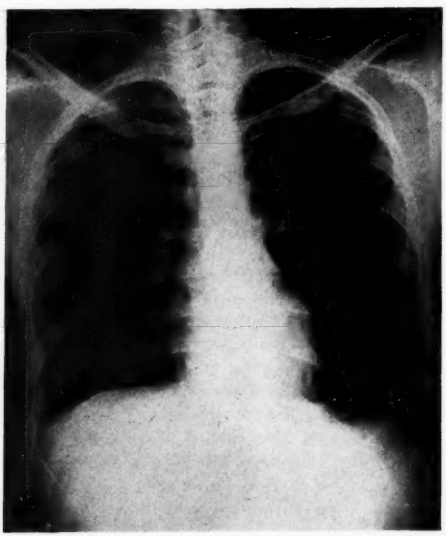

Fig. 3.—Posteroanterior film of chest Jan. 9.

placed over the site of repair and the defect in the mediastinal pleura closed loosely over the strip. Each twenty minutes during the procedure, tracheobronchial tree toilet was accomplished by the anesthesiologist, saline solution was placed in the pleural cavity, and the lung was fully inflated. A thoracostomy catheter was inserted and the chest wall hernia repaired as the thorax was closed.

Recovery was uneventful. The chest roentgenograms (Figs. 1, 2, and 3) depict the situation and the early result.

DEFINITIVE SURGICAL TECHNIQUE

The actual procedure conceived and utilized at the time of surgery is illustrated in Fig. 4. Actual ligation of a pleural cone could achieve the same end product as pictured in Fig. 5.

Also offered is the possibility of using a polyethylene catheter splint and, after the scarred ends of the duct are removed, fashioning a duct of mediastinal pleural flaps in the case of a long defect. The catheter could be brought out

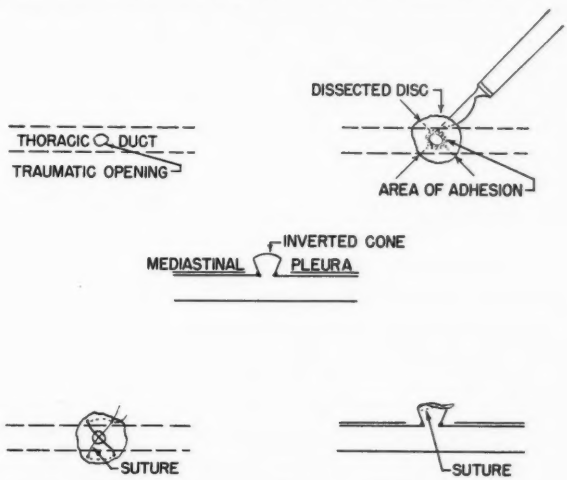

Fig. 4.

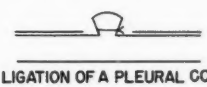

Fig. 5.

of the duct distally by a Witzel type of technique. Admittedly, the thoracic duct is quite small, but why could it not be repaired much as an extrahepatic biliary channel?

Apparently ductal ligation is relatively simple; however, we have been indoctrinated with the dictum of preservation of tissue including collaterals. We have read nothing of the ability of the thoracic duct or other lymphatic channels to recanalize.

CONCLUSIONS

1. Traumatic chylothorax should be kept in mind as a possibility in every case of pleural collection of fluid whether due to surgical or accidental trauma to the neck, the spine, the chest, or its contents.

2. Daily completely evacuant thoracentesis is essential to avoid serious respiratory and cardiac embarrassment. If seven days of such a regimen does not alleviate such embarrassment and leakage of chyle does not subside, we believe closed (tube) drainage with applied suction should be instituted. If seven days of the latter therapy does not produce definite progress, we believe in exploratory thoracotomy. If one is interested in preserving collaterals or is fearful of pressure rupture of the duct proximal to a ligature, a plastic repair may be feasible.

3. Bed rest may be considered essential to decrease the pumping action of respiration.

4. Nutrition is all-important. The patient should be weighed daily after thoracentesis, and intake and output faithfully recorded. Oral starvation should prevail. Intravenous alimentation with glucose, protein, and perhaps lipoids should cover losses. Further experimentation should be carried out with intravenous chyle and with the oral combination of fatty acids and glycerol.

5. The value of vagal stimulants to constrict the duct and drugs to paralyze the gastrointestinal tract, as well as gastrointestinal intubation, is questionable.

6. We have endeavored to describe a simple, practical plan to follow if one is faced with the care of a patient with traumatic chylothorax; and to indicate that prompt cure may be the reward for meeting the problem with boldness and simple mechanical measures.

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PROTECTIVE JACKET FOR THE POSTOPERATIVE PECTUS EXCAVATUM PATIENT

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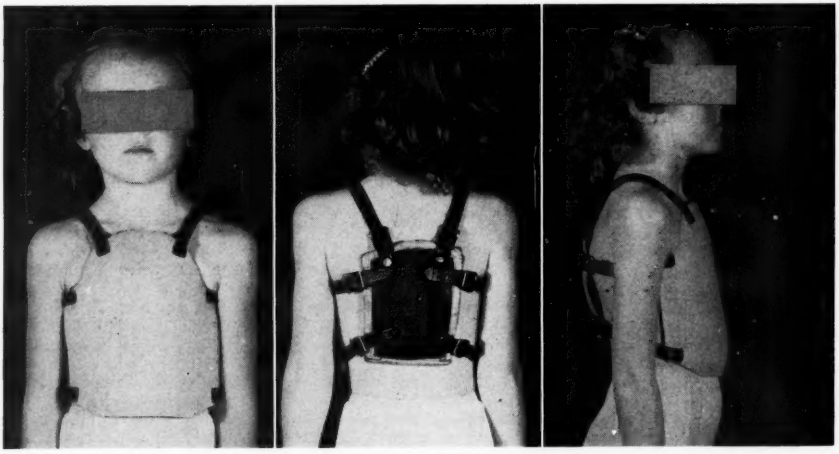
FOLLOWING radical operations for the correction of a funnel chest deformity with resultant instability of the anterior chest wall, great care must be taken to prevent injury to the thoracic viscera, particularly the heart. The patient must, of necessity, be very careful about his activity to prevent any accidental or sudden blows to the anterior chest wall. In the adult this is not particularly difficult, but in children this problem assumes great proportions. Child patients cannot be allowed to play with their friends for obvious reasons. If the child is kept in the house two to three months postoperatively there is always danger of his falling over a piece of furniture or suffering other injury which may result in damage to the anterior chest wall and heart.

This problem has been overcome to a great extent by the use of the anterior chest wall jacket shown in Fig. 1. This jacket, which costs approximately $40.00 for 5- to 8-year-old patients, is made of light weight plastic and fiber glass; it is adjustable and comfortable to the wearer at all times.†



A. *B.* *C.*

Fig. 1.—*A*, Anterior view. *B*, Posterior view. *C*, Lateral view.

After the jacket has served its purpose it is returned to the hospital for use on other patients. The average wearing time is approximately eight to ten weeks after operation. It is practically impossible for the patient to suffer injury to the anterior chest wall while the jacket is being worn.

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†Materials for this jacket are furnished by the Vernon-Benshoff Company, Pittsburgh 30, Pa.